

# ARIZONA MEDICINE

*Journal of ARIZONA MEDICAL ASSOCIATION*

VOL. 8, NO. 11



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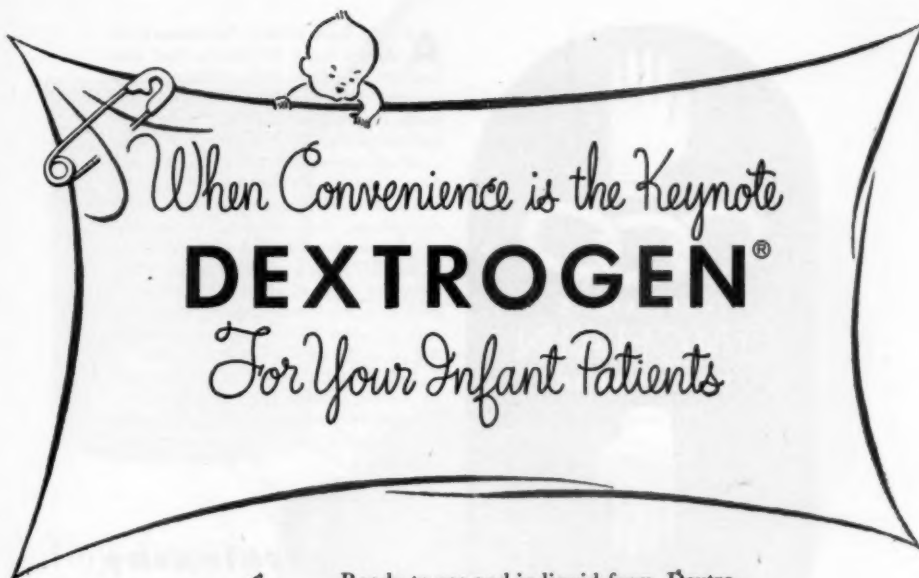
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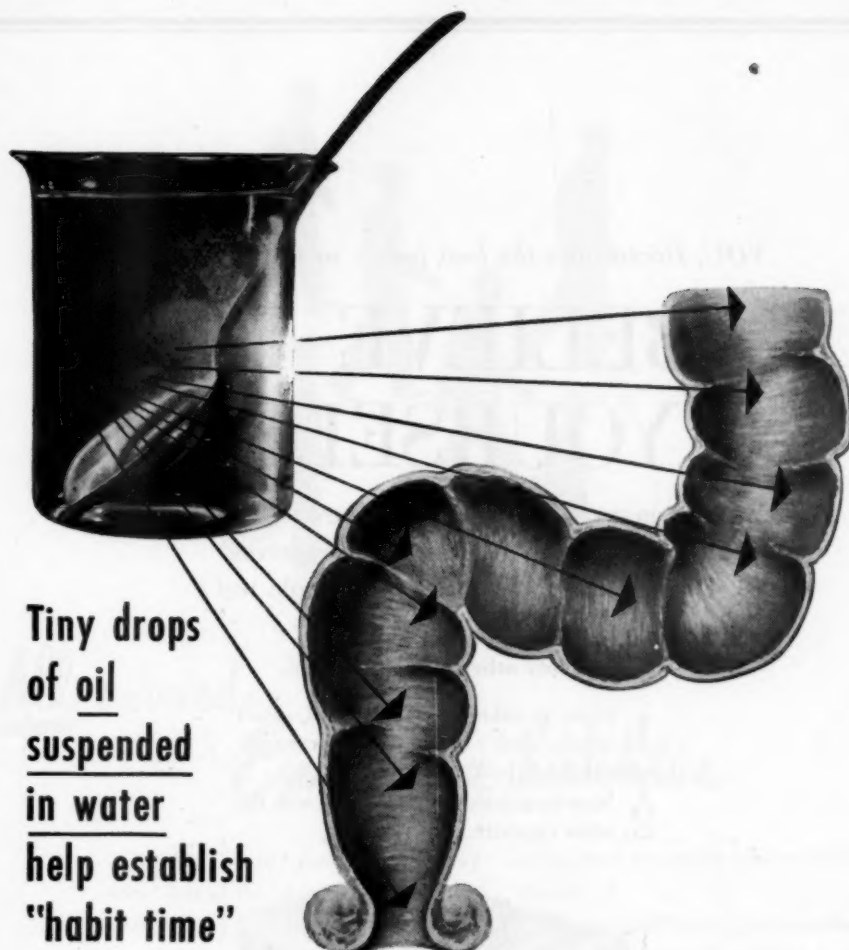
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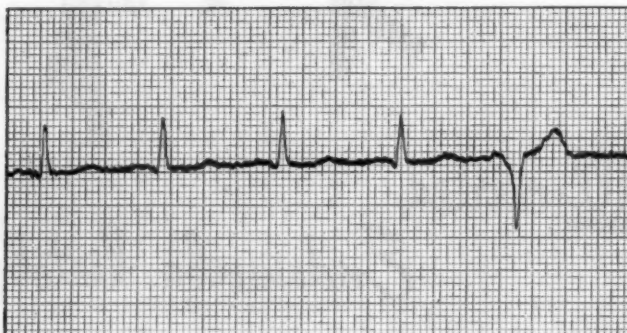
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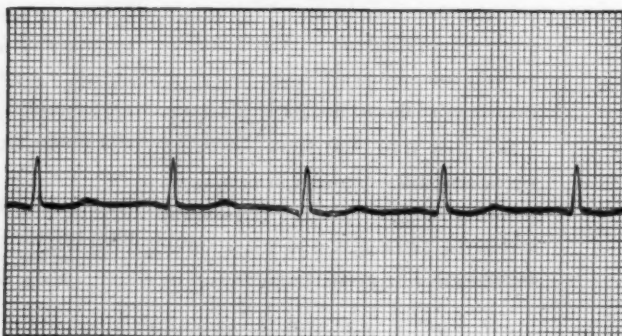
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#### BIBLIOGRAPHY

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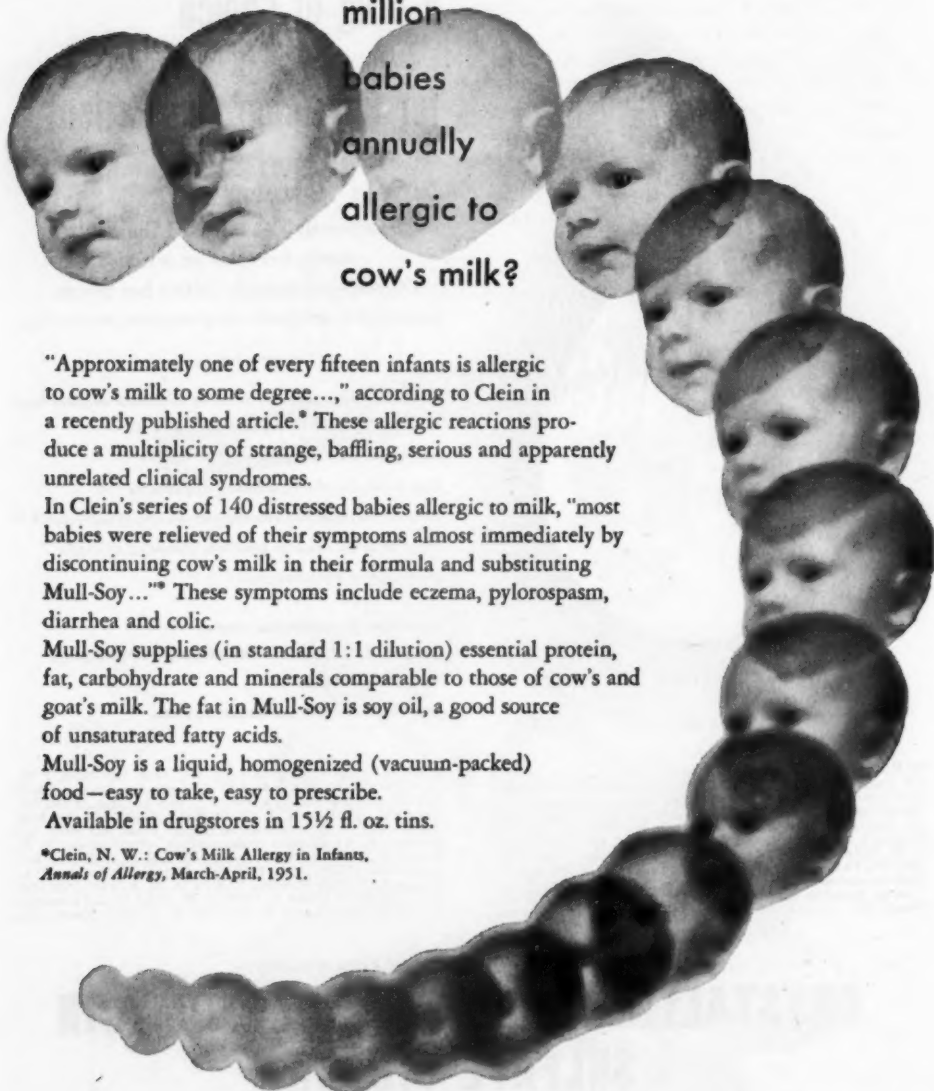
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\*Clein, N. W.: Cow's Milk Allergy in Infants, *Annals of Allergy*, March-April, 1951.

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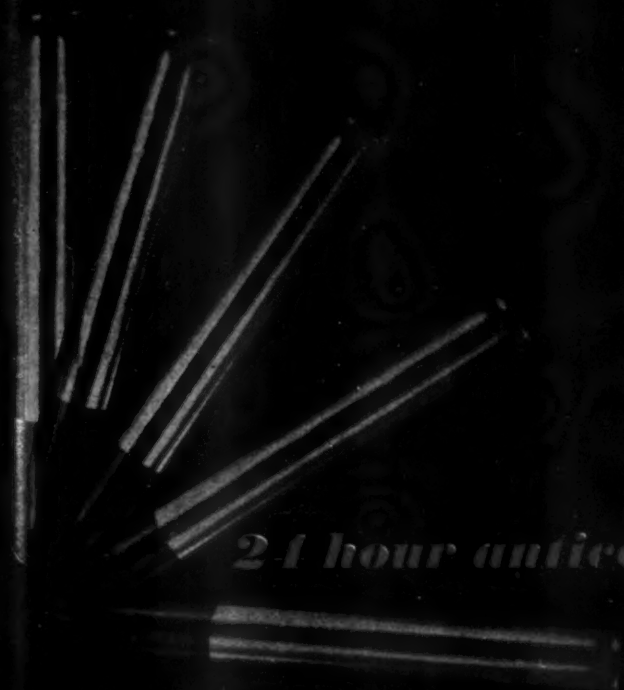
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### TABLETS

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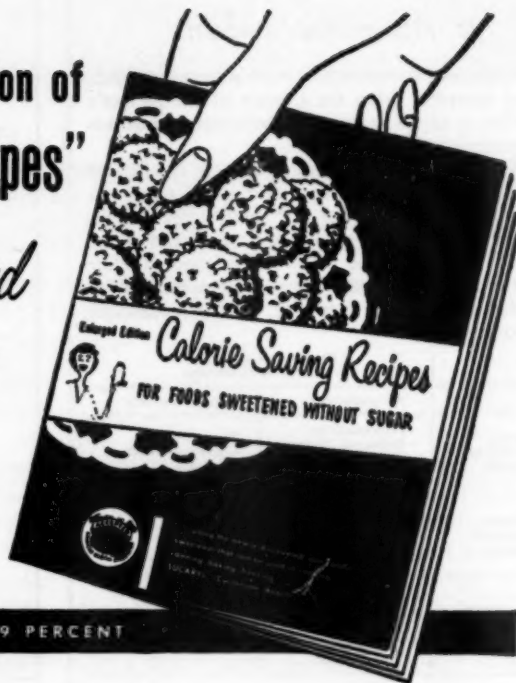
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## Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance.<sup>1,2</sup> Such states may involve any one of the organ systems or several at one time.<sup>3,4</sup> The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro- intestinal System	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio- vascular System	Rapid heart rate Peripheral vaso- constriction	Slow heart rate Vasodilatation
Functional Manifesta- tions	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references 3,4,5,6,7, given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure  
Body Temperature Variations  
Changing pulse rate  
Deviations in B. M. R.  
Exaggerated Cold Pressure Reflex  
Oculo-Cardiac Reflex Abnormalities  
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy\*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

\*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives, 8,9,10.

1. Ebaugh, F.: *Postgrad. Med.* 4: 208, 1948. 2. Wilbur, D.: *J.A.M.A.* 141: 1199, 1949. 3. Williams, E. and Carmichael, C.: *J. Nat'l. Med. Assoc.* 42: 32, 1950. 4. Goodman, L. and Gilman, A.: *The Pharmacological Basis of Therapeutics*, The Macmillan Co., 1941. 5. Katz, L. et al: *Ann. Int. Med.* 27: 261, 1947. 6. Weiss, E. et al: *Am. J. Psychiat.* 107: 264, 1950. 7. Alvarez, W.: *Chicago Med. Soc. Bulletin*, 581, 1950. 8. Rakoff, A.: *A Course in Practical Therapeutics*, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: *A Handbook of Psychiatry*, C. V. Mosby Co., 1945. 10. Harris, L.: *Canad. M.A.J.* 58: 251, 1948.

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# ARIZONA MEDICINE

*Journal of Arizona Medical Association*

VOL. 8, NO. 11



NOVEMBER, 1951

## Original ARTICLES

### SURGICAL TREATMENT OF ESOPHAGEAL LESIONS

DONALD L. PAULSON, M.D.

Dallas, Texas

Rapid advances have been made in the field of esophageal surgery during the last decade. It is now possible to perform satisfactory one stage procedures for a variety of esophageal lesions including congenital atresia with tracheoesophageal fistula, acquired tracheobronchoesophageal fistulae, acquired perforation or rupture of the esophagus, stricture, diverticula, cardiospasm, benign tumors and carcinoma. The aim of the surgical procedure utilized for any of these lesions is to establish esophagogastric continuity so that the patient can swallow his food in as normal a manner as possible. Ten years ago radical surgical procedures on the esophagus were either unheard of or looked upon with fear and discouragement because of the attendant high mortality and morbidity. Today it is possible to resect short segments of the esophagus and perform an end-to-end anastomosis, to resect any length of the esophagus including almost the entire esophagus and to perform a high intrathoracic or cervical esophagogastronomy, to perform an end-to-end anastomosis for a congenital esophageal atresia, to remove diverticula in a one stage operation and to perform various plastic procedures for stricture.

#### *Congenital Anomalies of the Esophagus*

The most common congenital anomaly consists of an atresia of the esophagus associated with a tracheoesophageal fistula. Either the atresia or tracheoesophageal fistula may be present alone in the absence of the other. Rarely, an esopha-

geal web may be present occluding the lumen. There may be an agenesis of the distal esophagus.

The diagnosis of congenital esophageal atresia associated with a tracheoesophageal fistula is easily made. The usual story is that of a newborn infant who breathes poorly and coughs and chokes on its own saliva or on attempts at feeding. In such an infant the passage of a small catheter into the upper esophagus will prove an obstruction. This may be visualized under the fluoroscope. One may readily introduce lipiodol into the proximal esophageal segment by means of a catheter (Fig. 1 A). Barium should never be used. The presence of air in the stomach and intestines when a blind upper segment is present proves a tracheoesophageal fistula. If air is not present in the stomach and intestines, but a blind upper esophageal segment has been demonstrated, one is dealing with either an atresia without the tracheoesophageal fistula or the distal segment of the esophagus is represented only by a cord. A tracheoesophageal fistula may be present in the absence of an atresia. In such an infant the catheter will pass normally into the stomach. However, instillation of lipiodol into the esophagus with the infant in the prone position will demonstrate a tracheoesophageal fistula.

Inasmuch as a congenital atresia with a tracheoesophageal fistula is incompatible with life, immediate surgery is indicated. The surgical treatment consists of first, division of the tracheoesophageal fistula and secondly, the establishment of normal continuity of the esophagus. This can usually be done in a one stage opera-

Read before Arizona Medical Association, Sixtieth Annual Meeting, Tucson, Arizona, May 1, 1951.





Figure 1A

tion in which an end to end anastomosis is performed between the esophageal segments after division of the tracheoesophageal fistula (Fig. 1 B). In those instances in which either the atresia or the tracheoesophageal fistula is present alone the problem is easily handled surgically. In a few instances the gap between the esophageal segments is so long as to preclude an end to end anastomosis or the distal segment may be represented only by a cord. In such instances, the tracheoesophageal fistula should be divided and the proximal esophagus brought out above the clavicle anteriorly as an esophagostomy. At a later date esophagogastric continuity can be re-established by using a jejunal transplant or performing a high intrathoracic or cervical esophagogastronomy.

Gastrostomy for feeding purposes should not be done until the tracheoesophageal fistula has been divided due to the danger or regurgitation of gastric contents into the tracheobronchial tree.

#### *Acquired Tracheoesophageal or Bronchoesophageal Fistulae*

Acquired tracheoesophageal or bronchoe-

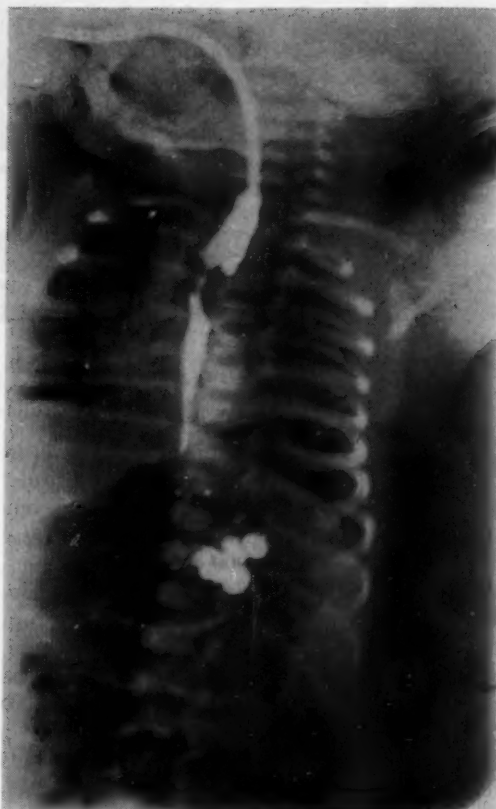


Figure 1B

sophageal fistulae may be secondary to trauma such as caustic burns of the esophagus, the erosion of tuberculous glands into these structures, or carcinoma. Those fistulae secondary to caustic burns or the erosion of tuberculous glands may be corrected surgically (Fig. 2B). If the fistula has not been present long enough to produce pulmonary suppuration, simple division of the fistula is usually sufficient to effect a cure. If a stricture of the esophagus is present a plastic procedure or resection of a portion of the esophagus may be necessary to establish normal esophagogastric continuity. If pulmonary suppuration has occurred due to a fistula of long standing, pulmonary resection may also be indicated. In those cases in which the fistula is secondary to the erosion of a tuberculous gland the patient usually gives a history of the expectoration of small hard particles which consist of pieces of a calcified lymph node. A simple division and repair of the fistula usually is sufficient in these cases.



Figure 2B

#### *Benign Acquired Stricture*

Acquired strictures of the esophagus are usually secondary to caustic burns, peptic ulceration, trauma, surgical procedures and tuberculous glands. The stricture may be partial or complete (Fig. 2C & 4A). Surgical treatment is indicated in those cases which do not respond well to the more conservative measures such as dilatation. Various plastic procedures, local excision with either end-to-end anastomosis or esophagogastrostomy, or esophagogastrostomy without excision may be carried out. In certain instances resection of the esophagus and the substitution of a piece of jejunum for it may be done.<sup>(1)</sup>

#### *Perforation or Rupture of the Esophagus*

Spontaneous perforation of the esophagus is not a rare condition judging by the frequency of reports recently made in the literature. Perforation of the esophagus may be spontaneous in a presumably normal esophagus, it may be secondary to trauma following the ingestion of a foreign body or a surgical procedure such as dilatation, or it may be due to perforation of a peptic ulcer. The esophagitis associated with a diaphragmatic hernia may antedate the perforation. The patient's history is very important in determining the cause of the perforation. The history of ingestion of a foreign body



Figure 2C

or operative manipulation will establish trauma as the cause. The previous history of symptoms of ulcer of the esophagus or of an esophagitis associated with an esophageal hiatal diaphragmatic hernia should make one suspicious of the underlying cause. In most of the cases of spontaneous perforation of the esophagus reported in the literature there has been no background in the history that would suggest disease of the esophagus.

Although the symptoms and findings differ according to the cause any esophageal perforation produces mediastinitis and not infrequently an empyema also. Prompt surgical attention is necessary in addition to the liberal use of various antibiotics. The diagnosis can readily be made on the basis of the history and the roentgen findings. These latter will reveal air or fluid within the mediastinum behind the heart or in the neck. A swallow of radiopaque oil with fluoroscopic observation may be demonstrate the esophageal perforation (Fig. 4B).

The treatment of esophageal perforation varies with the cause, the size of the perforation, and the duration. In any case the mediastinitis or empyema must be treated by drainage. If the perforation is seen early, whatever its cause may be, operative closure of the opening in the esophagus may save the patient a long and

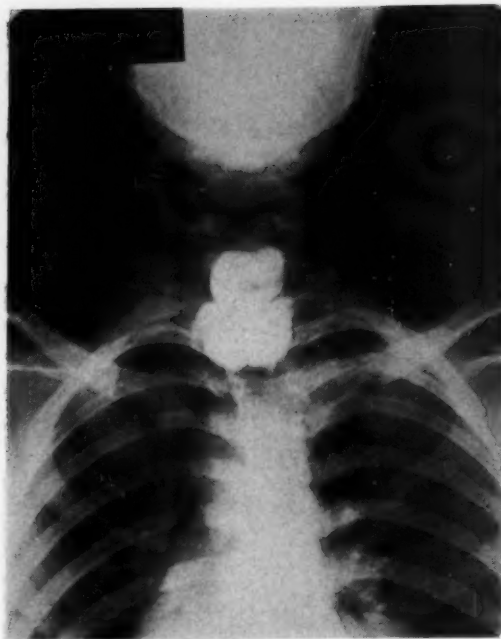


Figure 4A

severe illness.

Spontaneous perforation or rupture, presumably in a normal esophagus, usually occurs during vomiting, most frequently after a bout of alcohol ingestion. These perforations are usually very large and the mortality almost 100 per cent if treated conservatively. The treatment of choice consists of prompt thoracotomy, wide opening of the mediastinal pleura, closure of the esophageal defect, adequate drainage of the mediastinum and closure of the chest with intercostal tube drainage.<sup>(2)</sup> This procedure must be carried out in spite of the patient's poor condition. The large opening in the esophagus leads to a rapidly extending mediastinal infection which can only end fatally in a short period of time. Attempts to drain the local infection by mediastinotomy or pleural drainage during the acute stage have generally been unsuccessful in the case of spontaneous perforation in a presumably normal esophagus.

#### *Diverticula*

A diverticulum of the esophagus may be pharyngoesophageal or epiphrenic in location. The traction diverticula due to a tuberculous gland or trauma may be located anywhere along the extent of the esophagus. Diverticula vary as to size and extent but as time goes on they usually progressively enlarge.

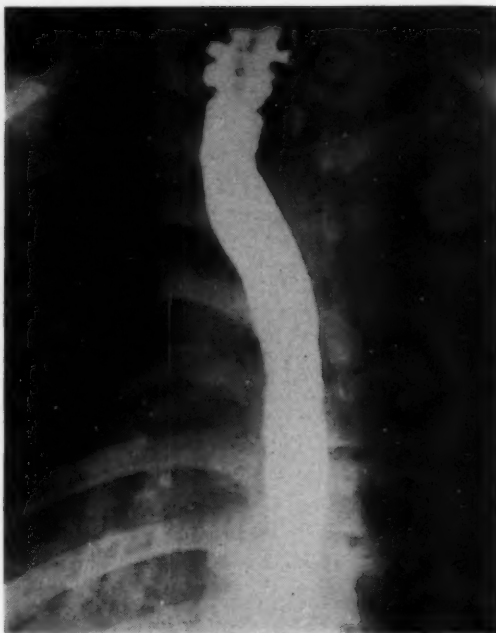


Figure 4B

The weight of the diverticulum and its contents pull it in a downward direction so that it narrows or compromises the normal esophageal passage. Its own opening progressively enlarges. A varying amount of inflammation occurs in the wall of the diverticulum and ulceration and perforation may occur as complications. One stage surgical removal can now be done with a high degree of success without fear of mediastinitis.<sup>(3)</sup>

#### *Cardiospasm*

In those cases of severe cardiospasm or achalasia of the esophagus in which repeated dilatation of the esophagus does not produce the desired results or in those cases in which pulmonary complications or severe anemia are complications, a plastic procedure at the esophago-gastric junction to enlarge the opening may be indicated and can be done transthoracically at a low risk.

#### *Benign Tumors*

Benign tumors of the esophagus are relatively uncommon. They may be divided into two main groups—the mucosal and the extramucosal tumors. The mucosal tumors are pedunculated and arise from the submucosa. Usually they are fibrolipomas or fibromyxomas covered by normal epithelium. The extramucosal tumors arise from the muscle of the wall of the esophagus.

gus and usually do not involve the mucosa. These are commonly leiomyomas.

Growth of these tumors is usually slow and therefore they may attain considerable size before giving rise to serious symptoms. Symptoms produced by these tumors are dysphagia, pain, regurgitation of food, cough and dyspnea. A pedunculated tumor may be regurgitated itself into the mouth. The diagnosis is readily made by the roentgenographic and esophagoscopy examination of the esophagus.

Although the indications for surgical treatment of benign tumors are not as urgent as for malignant lesions of the esophagus, surgical removal is indicated. These tumors are usually large when first seen, and therefore act as a mediastinal tumor by compressing the esophagus and the adjacent mediastinal organs. It is considered possible that these tumors may undergo malignant change. The pedunculated tumors may be readily removed through a cervical or transthoracic esophagotomy. The intramural tumors may be shelled out from the wall of the esophagus preserving the intact mucosa, the wall being reconstructed after removal of the tumor. In a few instances the mucosa will be damaged in the removal of the leiomyoma which may necessitate a resection and an anastomosis.

Small pedunculated tumors may be removed through the esophagoscope by means of a snare and cautery.

Esophageal cysts do occur and may be readily removed. They produce symptoms by pressure on the esophagus and adjacent mediastinal organs such as various parts of the tracheobronchial tree.

#### *Carcinoma of the Esophagus*

Cancer of the esophagus is of more frequent occurrence than are all malignant tumors of bone and is even more common than cancers of the lip, tongue, larynx or kidney. It constitutes about 4 per cent of the deaths in cancer of the esophagogastrintestinal tract in the United States (Gastric cancers 56%) (4) and 5 to 10 per cent of all carcinomas in males, being surpassed in frequency only by carcinoma of the stomach, lung and rectum. Until ten years ago, there was justifiable pessimism concerning esophageal cancer. Surgical procedures available were mutilating and were staged procedures. Very often the patient died from his cancer before the surgical plan of attack could be completed. The first successful resection and esophago-

gastric anastomosis for esophageal cancer was reported by Adams and Phemister in 1938.(5) During the ensuing ten years, this operation has been extended and modified so that now it can be applied in the treatment of carcinoma of the esophagus at all levels with the exception of the cervical segment. The morbidity and mortality of this surgical procedure have reached acceptable low levels. Although sufficient time has not elapsed for a statistical study of end results, reports which are available (6) suggest that carcinoma of the esophagus treated by radical resection will behave like carcinoma elsewhere in the body (lung and stomach) and that there will be enough cases of three to five year survival to justify the use of the operation in the hope of effecting a cure. Because of the pitiable plight of the patients who are unable to swallow because of complete esophageal obstruction, it is justifiable to make an effort to remove the cancer in every case, however hopeless the prognosis may seem. One of the major benefits of the operation is the restoration of esophago-gastric continuity and the ability of the patient to swallow food. Many such patients may live in relative comfort without any recurrence of dysphagia for as long as two or more years after resection and an esophagogastrotomy.

The obscurity of symptoms all too frequently results in a late diagnosis. Most patients coming to surgical treatment have had dysphagia for a period of six months or more. The medical profession as a whole must be alert to the possibility of esophageal cancer in order to make the diagnosis early. Any patient with dysphagia deserves careful roentgenographic and esophagoscopic investigation before any treatment is instituted. These two diagnostic procedures will readily reveal the presence of an esophageal carcinoma.

The surgical procedure utilized for a carcinoma of the esophagus varies according to the region where the tumor lies. The restoration of continuity after resection of a carcinoma in the cervical segment of the esophagus requires the substitution of a turned in portion of skin for the excised portion of esophagus. This may be accomplished by the method described by Wookey.(7) If the carcinoma is located in the upper fourth of the thoracic segment (in the superior mediastinum) a one stage esophageal resection with a cervical esophago-



gastrostomy is performed by means of two incisions. Through a left thoracotomy incision the entire esophagus from the base of the neck to the cardia is dissected free and resected. The thoracic incision is closed and a second incision is made over the lower cervical region anteriorly. The esophagus is dissected out through the incision, the fundus of the stomach being brought up into the cervical region from the chest, and the esophagogastric anastomosis is done within the neck above the level of the clavicle actually in the subpharyngeal region.

A carcinoma in the middle half of the esophagus (extending from the superior border of the aortic arch to the inferior pulmonary vein) is treated by esophageal resection and a high intrathoracic esophagogastrostomy. Whether the anastomosis is done superior or inferior to the aortic arch, depends on the location of the tumor. Any cancer which lies at least in part behind the aortic arch requires a high level of resection in order to remove the cancer adequately. Esophageal carcinoma characteristically extends submucosally so that wide resection is advisable in an effort to extirpate the disease.

A carcinoma in the lower fourth of the thoracic segment—that is that portion lying between the inferior pulmonary vein and the stomach—

is treated by resection of the esophagus and a portion of the stomach lying along the lesser curvature and a low intrathoracic esophagogastrostomy. Without pathological examination it may be difficult to determine the exact origin of the tumor whether it be esophagus or stomach. From a practical standpoint, however, the operative technique is the same in either case.

#### Summary

As a result of rapid advances in the field of esophageal surgery in the last decade, it is now possible to perform satisfactory one stage procedures for a variety of esophageal lesions including congenital atresia with tracheoesophageal fistula, acquired tracheoesophageal fistula, acquired stricture, perforation or rupture of the esophagus, diverticula, cardiospasm, benign tumors and carcinoma.

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### CHOLECYSTECTOMY WITHOUT DRAINAGE

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This is not a new subject. In 1906, Witzel(16) of the Berlin Clinic reported 500 cases of cholecystectomy without drainage and yet today, 40 odd years later, cholecystectomies are routinely drained in many hospitals. One looks back not so many years to the day when appendectomies, cesarean sections, gastric and intestinal operations were likewise routinely drained; whereas now, with a sense of progress, one notes that as a rule the abdomen is tightly closed in these cases. Gallbladder surgery does not enjoy this same progress, yet the proponents of non-drainage cite the following advantages of cholecystectomy without drainage, namely:

1. A cleaner wound with less chance of infection.

2. Less opportunity of inducing infection into the peritoneal cavity from the outside.
3. Fewer eviscerations.
4. Fewer herniae.
5. Avoidance of persistent sinus formation.
6. Less danger of bile leakage from the stump because of the tendency toward leakage is increased by pressure of the drain on the cystic stump preventing peritoneal agglutinins from covering and scaling off the stump.

The literature contains numerous reports of cholecystectomy without drainage; however, only those series in which mortality rates are given appear here:

Note the favorable mortality in the non-drained cases in this series.

In reviewing these cases, particular attention was paid to the incidence of bile peritonitis.

"Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statement and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration."



Name	Year	Without			Cases With		
		Drainage	Mortality	%	Drainage	Mortality	%
Abell	1927	391	8	2.04%	109	2	2.00%
Bailey	1939	129	0	0.00%			
Bettman	1942	306	5	1.63%	67	0	0.00%
Bottomley	1920	19	0	0.00%			
Coventry	1923	50	0	0.00%	30	4	13.33%
DeCoursey	1933	36	1	2.77%	300	20	6.66%
Fowler	1928	240	8	3.33%	543	15	2.76%
Mallet-Guy	1947	116	1	0.89%			
Verbrycke	1927	86	0	0.00%			
Willis	1917	35	1	2.86%	69	0	
Willis	1921	41	1	2.44%			
Witzel	1906	500	1	0.02%	(Bile peritonitis		
Total		1,949	26	1.34%	1,118	41	3.66%

All of the authors made a statement that no bile peritonitis occurred in these series except the single case of Witzel's. One may ask what becomes of the bile that forms in the undrained abdomen and the proponents of non-drainage argue that it does not form as seen in the re-operated cases.

#### 429 CHOLECYSTECTOMIES AT THE GENESEE HOSPITAL\*

\*Teaching Hospital Associated with the University of Rochester School of Medicine and Dentistry.

Four hundred twenty-nine cholecystectomies were performed at the Genesee Hospital from 1936 to 1946. Three hundred ninety eight cases were drained and 31 not drained.

Bile peritonitis developed in five cases resulting fatally in three instances, with a mortality rate of 60%. Two of these were in the 31 undrained cases and three were associated with common duct explorations in the drained cases. There were no instances of bile peritonitis in the drained cases when the common duct was not explored.

This series is striking in that non-drainage resulted in bile peritonitis in two out of 31 non-drained cases and in one it was fatal.

Because most cholecystectomies are drained in spite of theoretical advantages of non-drainage, it is felt that other surgeons in other institutions have had similar experience with bile peritonitis in undrained cases. It is probable that the ill effects of non-drainage do not regularly appear in the literature. The unexpected drainage of bile in the first few days following a simple cholecystectomy has often puzzled sur-

geons as to its cause. Unrecognized accessory bile ducts are incriminated. The proponents of drainage advise routine drainage to avoid an occasional fatality from bile peritonitis. Most of the older and more experienced surgeons will say that they did not drain until they lost such a case.

The following questionnaire was sent to a number of surgeons in scattered areas of the country:

1. Do you advise routine drainage of Morison's Pouch following cholecystectomy?
2. In your experience and the experience of your institution have you noticed an increased instance of bile peritonitis and complications in the undrained cases?
3. Would you care to further express ideas on the subject? *Replies appear below:*

Warren H. Cole: University of Illinois

1. "With few exceptions, I recommend and institute drainage following cholecystectomy.
2. "I have not had enough experience with closure without drainage to state whether or not this method would be followed occasionally by complications such as bile peritonitis. However, we have had one patient on whom a repair for a bile duct stricture was performed. Drainage was used but the wound around the drain was closed too tightly. This patient developed bile peritonitis, and we had to open the wound 24 hours after the operation; she ultimately died with numerous abscesses.
3. "Accordingly, I recommend drainage to protect the occasional patient. In perhaps

no more than one or 2%, will significant escape of bile from a cystic duct, aberrant hepatic duct, etc., take place."

George Crille, Jr.: Cleveland Clinic

1. Do you advise the routine drainage of Morison's Pouch following cholecystectomies? "Yes."
2. In your experience and the experience of your institution, have you noticed an increased instance of bile peritonitis and complications in the undrained cases? "Always drain."
3. "I have seen bile peritonitis or leakage when least expected. The appearance of bile from the drain gives warning of this. I always drain for two or three days."

Laurence S. Fallis: Henry Ford Hospital

(Referred by Roy D. McClure):

1. Do you advise the routine drainage of Morison's Pouch following cholecystectomies? "Yes, without exception."
2. "We cannot say that there have been increased instances of bile peritonitis in undrained cases because it is the practice in this hospital to drain patients. However, every year we have two or three cases of bile peritonitis that have been operated upon elsewhere and not drained."
3. "We readily admit that there are many instances, and perhaps a majority of the cases of clean dissection and removal of the gallbladder that the abdomen could be closed without drainage. However, we are unable to determine which case will give trouble, and therefore we advise drainage in all cases, since there is extreme variation in the amount of drainage in these patients. Furthermore, we have no instances on record in this hospital in which any complication could be directly attributed to the presence of the drain. We use the Penrose type of drain exclusively."

John H. Gibbon, Jr.: Jefferson Medical College

1. "I do advise routine drainage of Morison's Pouch following cholecystectomies."
2. "As we routinely drain, we have had no experience with undrained cases."
3. "I might say that we regard drainage with a soft filled cigarette drain as a simple precautionary measure which does not increase the length of convalescence and I can see no reason for abandoning this simple procedure."

Frank Glenn: The New York Hospital

1. "We routinely drain following cholecystectomy."

Henry N. Harkins: University of Washington School of Medicine

1. "I now advise routine drainage of Morison's Pouch following cholecystectomies. Several years ago, for a short time, I did not drain Morison's Pouch, but gave up this practice because I now work in so many hospitals that I cannot follow the cases as well as I could formerly. I had no disastrous results in the few cases where the pouch was not drained."
2. "In our experience, as already stated, we have seen no disastrous results, but we have only used the non-drainage method in a half a dozen or less cases."
3. "I am coming to the belief that when drainage is done, it should be done with a Chaffin tube arrangement, or by the sump drainage method of Babcock. Generally speaking, I use sump drainage, or Chaffin tube drainage in almost all cases where I am going to drain."

Emil F. Holman: Stanford University

1. Do you advise the routine drainage of Morison's Pouch following cholecystectomies? "Yes."
2. In your experience and the experience of your institution, have you noticed an increased instance of bile peritonitis and complications in the undrained cases? "Two deaths from non-drainage in 1922 perpetrated by another surgeon. This has forever prejudiced me against it!"

(Referred from Dr. Wangenstein):

Arnold J. Kremen: University of Minnesota

1. "It has been the practice in this clinic for some time to regularly drain Morison's Pouch following cholecystectomies."
2. "We have on several occasions seen collections of bile in the subhepatic space and even the development of subphrenic abscesses which I believe came about as a result of failure to drain following cholecystectomy."
3. "I have seen very few instances wherein drainage in this area providing the drain is brought out through a stab wound lateral to the incision, has ever caused any difficulty, and there are times when I think it has prevented postoperative complications."

## F. H. Lahey: Lahey Clinic

1. "We drain down to the foramen of Winslow in all cases and have never closed a cholecystectomy without drainage.
2. "I think that we have had two fatalities from bile peritonitis in the patients who had been closed without drainage who came here late, with bile leakage and with their abdomens filled with bile. These patients had had their gallbladders done elsewhere."

## Alton Ochsner: Ochsner Clinic

1. "We do not do routine drainage of Morison's Pouch or of the abdominal cavity following cholecystectomy. Drainage in our cases of cholecystectomy is dependent upon the ability to carefully cover the cystic duct stump with serosa and the presence or absence of exudation from the gallbladder bed. We believe that there is a likelihood of the cystic duct stump blowing off unless it can be covered with peritoneum which will permit the holding of fibrinous exudate around it which can become organized. Unless we can satisfactorily cover the cystic duct with peritoneum we always drain the peritoneal cavity and we also drain when there is a continued ooze from the gallbladder bed.
2. "We have not noticed any cases of bile peritonitis or complications in undrained cases when we adhere to this rule."

## Herman E. Pearse: Strong Memorial Hospital

1. Do you advise the routine drainage of Morison's Pouch following cholecystectomies? "Yes."

## Samuel J. Stobins: Genesee Hospital

"At the Genesee Hospital, Rochester, New York, it is the policy of the Surgical Service to drain Morison's Pouch following cholecystectomies on all Staff cases; drainage in private cases rests entirely with the attending surgeon."

## Waltman Walters: Mayo Clinic

1. Do you advise the routine drainage of Morison's Pouch following cholecystectomies? "Yes."

## DISCUSSIONS

Part of this paper was presented by one of the authors (CGF) at a staff meeting at the Genesee Hospital. Because of the two incidences of bile peritonitis in 31 undrained cases at that hospital, it was believed that this might

have been due to ill-chance rather than to the method. Many surgeons were privately questioned over a period of years and it became evident that other institutions had had similar results from non-drainage so that most surgeons had abandoned it in favor of drainage. It is felt the favorable data in the literature are due to selection, many of the ill results not being reported.

## SUMMARY

1. Theoretical advantage of non-drainage following cholecystectomy are given.

2. The mortality rates in non-drainage are collected from the literature.

3. Four hundred twenty-nine cholecystectomies at the Genesee Hospital, in the ten year period between 1936 and 1946, are reviewed. In 398 cases which were drained bile peritonitis developed but three times and all these were associated with common duct exploration. Thirty-one "ideal" cases were not drained and bile peritonitis developed two times in this series; one proved fatal.

4. Reports of a questionnaire sent to a number of surgeons in various parts of the country are presented.

5. In spite of theoretical advantages and the available data in the literature, it is felt that young surgeons should omit drainage following cholecystectomy with a great deal of temerity.

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## PERSONAL COMMUNICATION

Cole, W. H.; Crile, G. Jr.; Fallis, L. S.; Gibbon, J. H. Jr.; Glenn, F.; Harkins, H. N.; Holman, E. F.; Kremen, A. J.; Lahey, F. H.; Ochsner, A.; Pearse, H. E.; Stobins, S. J.; Walters, W.

## WHAT TO KNOW ABOUT CARCINOMA OF THE LUNG

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### 1. FREQUENCY:

Statistics regarding the frequency of cancer of the lung vary considerably between hospital and hospital depending upon whether the medical and surgical staffs are especially interested in it and through reputation attract a considerable number of patients of this type. Naturally, it is from those who have a special interest that most of the publications have come. I suspect, therefore, that they give a somewhat exaggerated picture of its frequency in relation to other forms of cancer. Certain it is, however, that it is one of the most frequent forms of malignant disease particularly in the male, occurring as it does, in a proportion of 6 or 7 males to one female, and as such, warrants the increasing attention that it is receiving. During the 10 year period ending 1946 in the Department of Pathology of the Toronto General Hospital the third most frequent site of primary cancer was the lung, being preceded in the list only by the stomach and large bowel.

### 2. ETIOLOGY:

Nothing definite is known regarding the cause of cancer of the lung. It has been attributed to the use of tar in road building but Boyd showed that it seemed to be more frequent in Winnipeg than Toronto. It has been blamed upon the inhalation of petrol fumes but seems to be as common in rural as well as urban communities. The belief that it is due to tobacco does not seem to have been well founded. Its supposed relation to previous inflammatory disease of the lung is probably due to inaccurate diagnoses. Until further evidence is produced we must regard the etiology as unproved.

### 3. PATHOLOGY:

The histopathology need not concern us. The important thing is that it arises in the bronchi and that it may appear anywhere in the bronchial tree. Seventy-five to 80% arise in the primary and secondary division of the bronchi, the remainder more peripherally. It spreads by way of the lymph vessels to the bronchial lymph nodes, mediastinal nodes and thence along the communicating nodes to the neck or abdomen. Widespread metastases through the blood

stream are unusually frequent. The cells probably gain entry to the blood stream via the thoracic duct or by direct invasion of veins. It is one of the most frequent sources of bone metastases. Spread to the brain through vertebral system of veins is common. Pleural effusion may be inflammatory in origin or due to extension of the cancer to the pleura. A blood-stained effusion is usually due to carcinoma. Like all other forms of cancer its rate of growth varies greatly. The most malignant forms kill the host in a few months; the occasional patient may survive for 4, 5, 6 or even 7 years without treatment.

### 4. SYMPTOMATOLOGY:

I believe that we have all laid too much stress upon the classical picture of cancer of the lung and in so doing made the diagnosis appear too easy. If the course of events proceeds according to what would be expected, the growth in a primary or secondary bronchus acts first as a foreign body and produces an irritating cough; next follows clear or whitish sputum from over secretion of bronchial glands, with ulceration, bleeding occurs and the sputum becomes streaked with bright red blood or occasionally gross hemoptysis is seen. When the lumen of the bronchus has been narrowed enough to interfere with the clearing of secretions distal to the area, infection occurs and the sputum becomes purulent. At about the same time atelectasis occurs in the segment of lung distal to the growth and bronchopneumonia develops. The patient has bouts of fever and if the parietal pleura is involved, pleural pain. There is often a deep-seated pain or discomfort in the chest quite apart from the pleural pain. Unfortunately this classical picture is no more to be depended upon than the classical picture of appendicitis. The important thing to remember are that the development of a cough, a change in the character of a cough blood in a sputum, a sense of pain or discomfort in the chest should not be disregarded. Recurrence of pneumonia in the same area of lung means bronchial obstruction and demands investigation. The appearance of a lung abscess or bronchiectasis in a patient of cancer age should be regarded as secondary to cancer of the bronchus until this has been



excluded. Growths which arise peripherally are unlikely to produce any symptoms in the early stages. There may be chest pain of varying character or, if the parietal pleura is involved, pleural pain. The patient who complains of feeling that there is something unusual in his chest should not be reassured because physical examination of the chest is negative without an x-ray being made.

#### 5. DIAGNOSIS:

As in all other disease, the most important thing is to be aware of the possibility of cancer of the lung. "What one knows, one sees." A careful history is of first importance.

Physical examination of the chest may or may not yield information of significance. The early lesion, whether it be in the larger bronchi or toward the periphery, is unlikely to give rise to any change in physical signs. When, as a result of the blocking of a main bronchus, a considerable area of atelectasis has developed, there will be a shift of the mediastinum toward the lesion with diminished resonance and air entry over the area and harsh breathing. There may or may not be rales. Peripheral growths must, as a rule, be very large to be recognized clinically. In the presence of extensive atelectasis the decreased movement, decreased size of the chest deeper intercostal spaces and increased obliquity of the ribs on the affected side may be obvious. If pleural fluid has developed it, of course, gives the usual signs. No importance should be attached to the absence of changes in the physical signs.

The presence of a Horner's syndrome, of enlarged lymph nodes in the neck and axilla and enlargement of the liver should always be looked for. If an enlarged node is found it should be removed for diagnosis. If the patient is hoarse the vocal cords should be examined. A paralyzed cord usually means that the case is inoperable. Complete immobility of one leaf of the diaphragm suggests involvement of the phrenic nerve and usually inoperability.

X-ray films of the chest provide the most important single method of examination. If the growth is peripheral in location it is readily seen. In a central lesion the first change is always an area of atelectasis with slight shift of the mediastinum and perhaps elevation or depression of the hilum and elevation of the diaphragm. More extensive changes are, of

course, easily recognized. Unfortunately, widening of the mediastinum may be the most prominent finding due to involvement of the mediastinal lymph nodes. A perfectly normal x-ray film may be found but is extremely rare even in early cases. Laminographs should often be done. Good bronchograms may be very helpful either through demonstration of a definite block in a bronchus or a filling defect in the lung parenchyma corresponding to the area of atelectasis. Both of these changes may be found in the absence of new growth and must be interpreted, therefore, in relation to other findings.

A bronchoscopic examination should be done if the history is suggestive even though x-ray has been normal. In most clinics only about 50% of the cases are yielding positive biopsies. If more patients present themselves at an earlier stage this percentage is likely to become still lower. Examination of bronchoscopic aspirates and sputum for cancer cells has proved useful in many clinics. If the clinical history is suspicious and x-ray findings are suggestive, thoracotomy should be advised even though the bronchoscopic findings are negative and no cells have been found in the sputum or aspirates. Recently several patients have been operated on whose lesion was found on routine x-ray surveys in whom all other examinations were negative and who had no associated symptoms.

#### 6. DIFFERENTIAL DIAGNOSIS:

Pneumonia, particularly of the atypical variety, since it is segmental in distribution and may be associated with some degree of atelectasis, may be most difficult to differentiate. Primary atypical pneumonia rarely persists longer than 6 weeks. In doubtful cases it may be necessary to "wait and see" for that length of time. The presence of a foreign body in the bronchus must be considered even in the absence of history. Lung abscess or bronchiectasis developing after middle life must be regarded as possibly secondary to carcinoma until proved otherwise. Hodgkin's Disease or Lymphosarcoma in the mediastinum may occlude the lumen of a bronchus and produce a secondary atelectasis and an x-ray picture quite indistinguishable from advanced cancer of the bronchus. When doubt arises a therapeutic trial of x-ray will usually solve the matter since the lymphomas are highly radiosensitive. Peripheral growths may be confused with any other type of primary growth as well as tuberculous.



Occasionally the doubt may be overcome by an aspiration biopsy but thoractomy is usually necessary. Bronchial adenomas can be distinguished from carcinoma only on biopsy. An adenoma rarely produces an isolated parenchymal shadow. In the absence of positive sputum, tuberculosis may occasionally present some difficulty.

#### 7. TREATMENT

Surgical removal offers the only hope of cure since these growths are highly resistant to radiation. Lobectomy may be justified occasionally but pneumonectomy is the procedure of choice. Sufficient evidence has now accumulated to prove that these growths can be cured by surgical removal. As in cancer elsewhere cure

depends on the type of disease and the time of diagnosis. Palliative pneumonectomy is worthwhile in some cases to free the patient to symptoms that arise from infection distal to the growth and in the hope of making his demise more comfortable. The prolongation of life that can be accomplished may be worthwhile but I do not believe that a mere extension of life for a few months is in itself to be considered of great value.

Radiotherapy may have some palliative effect. The prolongation of life claimed by radiotherapists must be assessed with great care since some patients live so long without therapy of any sort. With improvement in technique it is worth further trial in inoperable cases.

### UNUSUAL VARIETIES OF PNEUMONIA AND PULMONARY ABSCESS

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It is the intention of this paper to point out how completely dangerous it may be to accept as satisfactory diagnosis such common nomenclature as abscess of the lung, atypical or viral pneumonia, and unresolved pneumonia.

Most of these terms come from the radiologists, and we are sometimes content to accept such a roentgenologic diagnosis without ado and immediately proceed to the use of the antibiotic with which we are currently infatuated. Prior to the days of roentgenography it was the custom to diagnose about everything that was associated with a high fever, pleuritic pain, cough and leucocytosis as pneumonia. If there were no satisfactory physical findings, it was influenza. The roentgenologists forced upon us a rather more accurate morphologic consideration of infection of the lung; they showed that disease was not always lobar or bronchopneumonic but might be segmental; that what we thought was pneumonia was often-times the inflammation of an abscess in which necrosis had not yet occurred.

Then came the first—perhaps the greatest—of the antibiotics, penicillin, and a new classification sprang up among clinicians. There were just two kinds of pneumonia then: those which responded to penicillin and those which didn't. In many places this simple classification seems to persist, though the multiplication of potent antimicrobial agents has tended to keep the

clinician somewhat more alert. In a sense, though, the classification is still somewhat the same, only a little broader: now there are infections that respond to penicillin; those that do not, but respond to some of the others; those that fail to respond to any antibiotic.

It is against such an ultra-empiric approach to disease that these remarks are delivered. There is and can be but one satisfactory approach to the problem of pulmonary infection and that approach is an etiologic one. The cases about to be shown are demonstrations of the necessity of pertinacious investigation. In this discussion it is fair to assume that two important differentials already have been carefully considered—as they should be in every instance in which a report of unresolved pneumonia, viral pneumonitis or abscess has been offered by the roentgenologist. Those two conditions are familiar and have been mentioned often: they are tuberculosis and carcinoma. Until those two diseases have been excluded, a beginning cannot be made on a pulmonary diagnosis.

But even after carcinoma and tuberculosis seem adequately excluded, there remain many other diseases that call for precise etiologic study. It is with these that this discussion deals. Just what percentage of pulmonary infection they represent is uncertain. These, of course, are called from many admissions. It probably is safe to estimate that such conditions

represent about 1% of all admissions for acute infections of the lung.

Several conditions are not represented since in the area from which these patients come there is a relatively low incidence of infestations, there is no mining and relatively slight industrial hazard and there is comparative freedom from the more exotic tropical diseases. In addition, but not included, are a number of pulmonary infections in which the most exhaustive efforts, including on occasion lung biopsy, have failed to produce a satisfactory answer.

In the majority of these unusual cases of pneumonia it is evident that they represent pulmonary complications or localizations of the less familiar infectious diseases. In a few cases the pneumonia is the result of some comparatively infrequent affection of the lung itself, while in others the infiltration of the lung may represent abnormal reaction or prior morphologic alteration of the tissue.

Table I presents a classification of pneumonias which has been found to be of value in approaching the problem of a baffling pulmonary infection.

Before proceeding into the presentation of individual cases, two further warnings should be added. So-called post-operative pneumonia or ether pneumonia, which characteristically is manifest about the third day is not primarily an infection, but is the result of bronchial obstruction. Although the condition responds to penicillin, the use of an antibiotic confuses the issue. The important step is the reopening of the occluded bronchus.

The other warning is to be on the alert for pulmonary infarction in the healthy, active individual. The roentgenologist in many instances will be unable to differentiate a subsegmental pneumonia from an infarct and physical examination not infrequently fails to reveal any evidence of peripheral venous disorders. Points of some aid in the differential diagnosis are the character of the sputum which will be mentioned later, the very dramatic onset, the relatively low temperature and leucocyte count and the early appearance of a sterile, hemorrhagic effusion (frequently seen only in lateral film). Jaundice seems not to develop unless preexisting hepatic disease is present and the electrocardiogram practically never affords a clue. A roentgenographic clue of great importance is the conversion, often within three or four days, of

the original consolidation into horizontal linear shadows which may persist for some days. Almost never does the postero-anterior or lateral film reveal a pyramidal or conical shadow.

The following cases of pneumonia illustrate varieties of unusual pulmonary disease. There are of course a group of notable omissions. Primary coccidioidal infiltrations are not encountered in the Dallas area; no proven case of infectious mononucleosis with pneumonic infiltration is included; strongyloides infestations of the lung, which seems to have been familiar to those in Burmese and Indian theatres, is missing.

TABLE I

Pneumonia of bacterial etiology.
Primary atypical pneumonia.
Pneumonia due to specific viruses.
Pneumonia of Rickettsial origin.
Pneumonia due to higher organisms.
Infections due to yeasts and molds.
Loeffler's syndrome.
Pneumonias resulting from systemic diseases.
Infiltrations associated with malignant diseases.
Pneumonia due to the inhalation of toxic gases and fumes.
Pneumonia due to inert foreign matter.

#### REPORT OF CASES

CASE 1. The patient was a white male of early middle age who entered the hospital with the complaint of fever, malaise, cough, headache and generalized aching. Laboratory studies were of note mainly for their lack of information. Bronchoscopy indicated a somewhat fixed and rigid intermediate bronchus on the left, but secretions were negative on Papanicolaou staining. Fever responded slowly and gradually over a period of time and it is impossible to state that any antibiotic produced a definite effect. Since the shadow persisted unchanged over a period of some time, it was considered safer to explore for carcinoma than to accept negative findings as being exclusive. The tissue grossly revealed a chronic organising pneumonia; histologically it was said to be compatible with the infiltrations characteristic of Q-fever. Serologic studies for that disease which had been weakly positive shortly before surgical exploration subsequently became much more strongly positive. (1, 2).

CASE 2. This patient, a young white male, entered the hospital with high fever and considerable malaise. On his skin there were a few small vesicles, which after two days became

much more numerous and took on a hemorrhagic character. Four days later the temperature began to subside and the patient was entirely well on the sixth day. His two children had recently been confined with known chickenpox. (10).

CASE 3. The patient, a farmer in late middle age, had been working in the fields during intensely hot weather. He suddenly became intensely ill with high fever, photophobia, marked muscular pain and cough. When he entered the hospital his temperature ranged around 103-104°, his entire body was quite tender to touch and his cough was productive of dark blood. Six or eight days later jaundice appeared. Chest taps resulted in bloody fluid. Dyspnea became intense in spite of repeated taps and oxygen. By the twelfth hospital day cyanosis and dyspnea were severe, there was intense jaundice, marked cylindruria and albuminuria and rapidly rising urea. All the common agglutination tests were negative and although the specific etiologic agent was never demonstrated we feel quite safe in presenting him as a case of Weil's Disease.

CASE 4. A middle-aged white female had been ill at home for about a week. There had been considerable cough, some shortness of breath and wheezing and a moderate degree of fever. In spite of aureomycin, she had become worse and her attending physician noted an increase in an area of consolidation. Chest film revealed a somewhat irregular consolidation. In the hospital her course was marked by temperature as high as 103, a moderate leucocytosis with persistent eosinophilia of varying degree and not always constant. The only organism recovered from the sputum by repeated culture was streptococcus viridans. When she was first placed on penicillin there was a short period of improvement lasting about four days, at the end of which the temperature which had nearly reached normal began to rise steadily. No combination of penicillin with any other antibiotic proved of advantage. When penicillin was increased there was always a short period of response. For six weeks the picture remained essentially unchanged, the patient at all times showing rather marked dyspnea, a slight violaceous cyanosis and experiencing some pleuritic pain from time to time. At last, for the want of any other satisfactory therapy, it was decided to place her on doses

of penicillin in the range ordinarily used for subacute bacterial endocarditis. On this regimen her temperature fell rapidly to normal and remained so thereafter. Some three weeks after her discharge and about nine weeks after the onset of her illness, resolution of the lower lobe pneumonia was essentially complete, when suddenly a new patch appeared in the left upper lobe. This migratory behavior seems to justify our diagnosis of Loeffler's syndrome, since eosinophilia was fairly clear throughout. If this is correct, it is somewhat unusual to find such a relationship with what was apparently a pneumonia due to streptococcus viridans. Since that time, for a period of nearly two years, the patient has been quite well.

Acute abscess of the lung is usually dismissed simply with that diagnosis. Most abscesses are of the usual mixed, anaerobic variety, the predominant organism probably being an anaerobic streptococcus. The mode of onset is usually sudden, often following a period of unconsciousness from whatever cause. The patient presents within a matter of hours the picture of severe pulmonary infection with hyperpyrexia, pleuritic pain, severe cough, dyspnea and cyanosis. Clubbing of the fingers may develop within as short a time as seven days in a few severe and large abscesses. Films up to the third or fourth day rarely reveal evidence of cavity formation, but are suggestive of abscess by the predilection of that disease for the dorsal segment of the lower lobe and for the axillary portion of the upper. From about the fifth day on—assuming treatment has been delayed for any reason—the cough becomes productive of foul sputum and roentgenograms of the chest reveal rapidly developing rarefaction, with or without fluid level.

Under present conditions many of these are aborted in the pneumonic, precavitary stage and undergo complete resolution. Either by reason of inadequate treatment or delayed treatment a certain number of patients proceeds to a modified variety of the once familiar picture. In many cases due to antimicrobial therapy, the sputum never becomes frankly putrid.

We are now particularly concerned with those patients who, having received adequate treatment, have persistent cough, sputum, fever and roentgen evidence of cavity. Here as in the case of the pneumonias one must always be sure



that tuberculosis and carcinoma have been well excluded. But even after these two prominent possibilities are clearly put aside, there remain certain other necrotizing diseases of the lungs which have to be considered. There are, of course, abscesses due to unusual organisms, mallei, bacteroides, klebsiella, and blastomycosis. Of these we have no examples, yet such diseases are probably not uncommon in certain areas. Other etiologic types are notable for their roentgen similarity to ordinary anaerobic abscess, for their protracted course and for their almost complete resistance to treatment with any antibiotic. Certain characteristics of their behavior, however, might have offered clues at earlier dates, while others even in retrospect cannot well be differentiated by any method from simple abscess.

CASE 1. A white male of early middle age went to a local tuberculosis control unit complaining of fever, cough, expectoration, malaise, loss of weight and weakness. A chest film revealed at that time (some four weeks prior) essentially the same situation evident in the slide. Although a sputum was negative upon a single examination, he was regarded as having tuberculosis and sent to the city sanatorium. He was moderately febrile, there was persistent leucocytosis, and continued cough with the expectoration of considerable amounts of yellow or greyish sputum which was always negative for tubercle bacilli. Carcinoma was included in the differential diagnosis, but bronchoscopy with cytologic study of secretions was negative. He was then considered to have an abscess and was placed on considerable doses of antibiotics, first one at a time and then in combination, all with little or no result. Numerous agglutination tests failed to afford any useful evidence. Finally wet mounts of the sputum revealed motile *endamoeba histolytica*, and following this cysts were found in the stool. He was treated with emetine, diiodoquin and chloroquin, the last of which produced dramatic results. The clue in this patient's case, so far as there was one, was the presence of a very slightly enlarged and moderately tender liver. Jaundice was absent and liver function tests were normal. The patient denied any symptoms suggestive of amebic colitis. (7, 8).

This case is of more than passing interest in that abscess of the lung, non-contiguous with the hepatic process is quite uncommon and

that the result with chloroquin treatment was so extremely successful. The outcome of such cases as given by Ochsner and DeBakey (6) has been marked by a high fatality rate (prior to availability of chloroquin).

CASE 2. A young colored man, known to be alcoholic, appeared at the admitting office with a temperature near 105. He was clouded mentally and at least a major part of the history had to be obtained from relatives, who stated that about two or three weeks earlier he had begun to have severe pain in the abdomen and flank, experience fever and chills, and to complain of headache. The patient was severely and acutely ill with moderate dyspnea and questionable cyanosis. Major guarding seemed to be present over the right upper quadrant. There was thought to be mild jaundice, which was confirmed by the icteric index. The day following pain shifted to the left flank and there was an increase of dyspnea, definite cyanosis and very considerable splinted cough. Two days later the cough became productive of dark red to brown, moderately thick blood. Mucus and pus were almost totally wanting from the sputum, which upon repeated examination for every type of pathogen, was negative. The patient remained dangerously ill for a period of some two weeks, at the end of which time his temperature began gradually to subside, his consciousness to clear and his cough and chest pain to diminish. He received in very considerable quantity every antibiotic available and the decline in temperature seemed to coincide with penicillin up to 3 million units per day. The chest film showed gradual clearing although the cavity remained, undergoing frequent changes in size. At the end of another four weeks the patient appeared clinically to be doing quite well and consideration of some method of procedure against the residual cavity was being taken when he suddenly sat up in bed one day, gasped and died. Autopsy revealed that the cause of death was a pulmonary embolus and that the abscess was in fact an acute, non-suppurative necrotic process involving what was originally an infarct. Acute and chronic cholecystitis was also discovered.

While this possibility was considered early in the differential diagnosis it never received the attention due it. That attitude resulted from the fact that while acute necrosis and cavity formation in infarcts is mentioned the condition

seemsto be an extremely unusual one. At no time was there evidence of circulatory disorder in the legs. Finally the history and the picture as presented at admission were strongly suggestive of an acute infectious disease in the background and Weil's Disease was rather strongly entertained for a time. If a clue existed in this case, aside from the negativity of all bacteriologic and serologic studies, it was in the character of the sputum. The jaundice, which otherwise might have helped, seemed to go better with acute disease involving the liver or gall-bladder for which there were good anamnestic and physical grounds.

CASE III. A white male in the fourth decade entered the hospital complaining of chest pain, shortness of breath and fever. Physical examination presented the usual signs of pneumothorax but otherwise was non-contributory. There was leucocytosis with a sharp shift to the left. X-ray revealed a hydro-pneumothorax, while within the lung itself was a rounded, very thick-walled cavity which seemed in the standard projection to lie just beneath the pleura. Since the patient had seen service during the war in the West Texas and in the Arizona area, a coccidioidin skin test was done and the reaction was positive. Culture of both sputum and plueral exudate resulted in ready growth of coccidioides. The patient prior to admission had had a chronic productive cough of some years duration, the sputum being rather consistently yellow in color, but non-foul.

CASE IV. A white male in the latter part of the sixth decade stated that for some three or four weeks previously he had noted fever, cough and sputum of unpleasant taste. On one occasion there had been hemoptysis of perhaps as much as 90 cc of fresh blood. The patient appeared acutely and chronically ill and the greenish, muco-purulent sputum was somewhat offensive in odor. Rhonchi were heard near the hilum and beyond this there were dullness, diminished breath sounds and a few rales. Bronchoscopy revealed no visible pathologic process other than a reddened mucosa. Secretions studied for cellular morphology were reported as showing a few atypical cells, but no definite psoitive diagnosis was possible. The patient was subjected to exploratory thoracotomy after preparation with antibiotics. Grossly this was thought to be an abscess only, but dissection revealed a small tumor, which proved to be car-

cinoma, in one of the branch bronchi.

It is worth emphasizing that in the consideration of a patient in middle life who presents the picture of an abscess, it is of the utmost importance to secure a thorough history as to the mode of onset. In the event of a gradual onset, with a sputum which becomes foul only after some period of time, carcinoma is always a strong probability.

CASE V. The patient, a farmer from the eastern edge of Texas, entered the hospital in a very acutely ill condition. He had been treated for a short period of time at his home by his local physician but when penicillin had failed to result in any improvement in his cough he was referred to the hospital. It was learned further that the patient had had an abrupt onset with a chill and high fever some ten days before, that there had been some chest pain although it was not of a violent character, and that cough had begun a day or two later and had been productive of mucoid or mucopurulent sputum which at times contained both bright red and dark brown blood. The leucocyte count was elevated and there was a strong left shift. Sputum studies were not immediately revelatory and the routine agglutinations at the time of admission were not altered to a significant degree. Blood cultures yielded no additional information. Since the patient apparently had received adequate doses of penicillin he was placed on a combination of one of the other antibiotics with streptomycin. On this therapy over a period of some days his temperature gradually subsided and significant clearing of the consolidation occurred. As the patient became better able to cooperate it was learned that he frequently went hunting in the woods and had killed and skinned both squirrels and rabbits. A repetition of the agglutination studies for tularemia was strongly positive. This case is of unusual interest for although pulmonary manifestations of tularemia have frequently been reported and although one report mentions the formation of an abscess this is apparently the first case of tularemic abscess whose film has been reproduced. It is noteworthy that there is no obvious feature which would differentiate it from any other abscess so far as the film is concerned.

There has been presented today a group of acute and subacute diseases of the lung which admittedly are rather rare. The purpose of this



presentation has been to emphasize the importance of suspicion of one's own diagnosis and study. There is probably not one of this entire group of patients who might not have been correctly diagnosed earlier had a level of suspicion been sufficiently great.

For those who have studied these patients during their illness—they have seen demonstrations of the ultimate fallibility of all laboratory techniques. Roentgenographically, these abscesses and these pneumonias were not distinctive. From the standpoint of the clinical laboratory all usual studies were contributory only indirectly. In fact, until the suspicion of the correct diagnosis had been entertained and the laboratory was able ultimately to confirm these suspicions all valued tests merely served for exclusion.

On the other hand what has been demonstrated is the importance of searching clinical thought and study. In one instance the occupation, in another the habits, in a third the kind of sputum, in a fourth an apparently unrelated hepatic tenderness, in a fifth an evanescent rash contained the luminescent key to the problem.

What all this seems to point to is that the competent clinician must have constantly in mind the patterns of behavior of all the more common varieties of pneumonia and abscess and must cultivate an acute sensitivity to the abnormal behavior of a disease process. He must bear constantly in mind that the film of the chest may reveal only a chance localization of a generalized disease and must avoid the pitfall of mistaking a part for the whole. And above all else the clinician must remember that a careful history, a good physical examination and some close observation will lead more frequently to correct diagnosis than a tremendous multiplication of laboratory tests.

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## BASIC Science SEMINAR

### THE PHYSIOLOGY OF CELLULAR RESPIRATION, INCLUDING CARBON DIOXIDE AND OXYGEN EXCHANGE

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#### INTRODUCTION

Cellular respiration is defined by Reid (1) as those biological processes and cellular mechanisms by which the cell converts the bound radiant energy of the sun, stored in foodstuffs, to free utilizable biotic energy, thereby making possible cellular activity and even cellular existence. At once this definition limits the present discussion to those metabolic processes taking place within the cell, and excludes the more familiar phase of oxygen and carbon dioxide exchange in the lungs and extra-cellular fluids. A discussion of the specialized oxygen and carbon dioxide carrying cells of the blood, the erythrocytes, is also eliminated because these cells serve primarily in gaseous transport. Instead, it is the plan of this paper to take up those metabolic processes that are intimately

concerned with the metabolism of cellular contents, and to show some of the fascinating and complex mechanisms by which intracellular respiration takes place.

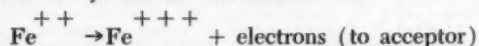
In order to discuss this topic, it is necessary for one to visualize the individual cell, surrounded by its cell membrane, and containing perhaps hundreds of complex interacting enzymes, catalyzing chemical reactions of foodstuffs or metabolites. This cell in turn must be thought of as part of a coordinated group of cells that takes part in the formation of a tissue and an organ. The problem of supplying these groups of cells with foodstuffs, oxygen, water, and of disposing of carbon dioxide and waste products is complex almost beyond comprehension. The physiology of cellular respiration concerns itself with the sum total of those chem-

ical reactions that have to do with the breakdown of cell metabolites for the release of energy required for cell work. Fundamentally these reactions are oxidations.

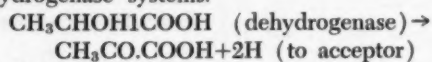
#### GENERAL PRINCIPLES OF BIOLOGICAL OXIDATION

In any chemical reaction, oxidation can be carried out by the addition of oxygen to a substance, by the removal of hydrogen, or by the withdrawal of electrons. In all three it is the loss of one or more electrons from the substance being oxidized that characterizes the oxidation reaction. In cellular or biological systems, four general types of chemical reactions may be recognized as oxidation reactions (2):

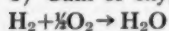
1) Loss of electrons, e.g. as found in the iron atom of cytochrome molecules.



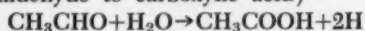
2) Loss of hydrogen, e. g. as in certain dehydrogenase systems.



3) Gain of oxygen



4) Addition of water with loss of hydrogen (aldehyde to carboxylic acid)



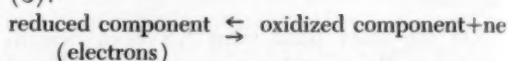
In reduction on the other hand there may be a loss of oxygen from a substance, or the addition of hydrogen, but fundamentally it is the gain of electrons by the substance being reduced that characterizes the reduction reaction. Since oxidation of one substance is invariably accompanied by reduction of another substance, an oxidation-reduction reaction implies a loss of electrons by the substance being oxidized and a gain in electrons by another substance able to accept electrons and undergo reduction. In living cells when molecular oxygen derived from the atmosphere is the ultimate hydrogen acceptor, the process is called respiration.

With the majority of known physiological metabolites, primary oxidation is actually accomplished by the removal of two hydrogen atoms, catalysed by specific cellular enzymes known as dehydrogenases. In a few instances the liberated hydrogen combines directly with molecular oxygen to form hydrogen peroxide and then water, but in the majority of cases the liberated hydrogen atoms or their electron equivalents are transported to molecular oxygen

by a series of reversible oxidation-reduction reactions. In the latter, oxygen appears only in the final stages of a chain of respiratory reactions, and then only in the form of water as the result of its reduction by hydrogen. In no known instance does oxygen combine directly with carbon to form carbon dioxide. Instead, carbon dioxide is extracted through the splitting out of carboxyl groups from metabolites by a process known as decarboxylation.

#### OXIDATION-REDUCTION SYSTEMS

An oxidation-reduction (O-R) system in simplest terms consists of two chemically related compounds which are capable of reversible transformation one into the other by the exchange of one or more electrons. For example, any O-R system can be represented as follows (3):



where (n) is the number of electrons transferred. The oxidizing or reducing power of such a system can be related to other similar O-R systems by comparison with a standard O-R system such as the hydrogen electrode. Adjustments are made for the fact that the hydrogen electrode is standardized at pH 0, whereas physiological systems must be measured at neutrality (pH 7). O-R systems with more negative potentials are more reducing than those with more positive potentials. For example, the hydrogen electrode with a potential of  $-0.421\text{v}$  at pH 7, represents a system with great reducing power, while the oxygen electrode with a potential of  $+0.810\text{v}$  at pH 7 represents an opposite extreme of a system with great oxidizing intensity. O-R systems must be thermodynamically reversible so that a small change in the equilibrium of the solution must be able to convert one of the components into the other. The significance of O-R potential studies is that they provide a method of predicting the possibility of a reaction between two O-R systems, and that they give the sequence in which the systems will carry electrons. O-R potentials also predict the amount of free energy able to be released at a particular stage in a series of multiple O-R reactions. Dehydrogenases, co-enzymes, and cytochromes take part in cellular O-R systems, each with an O-R potential, and make possible cellular respiration.

#### A GENERAL OUTLINE OF CELLULAR OXIDATION

The chemical reactions found in cells obey the same thermodynamic principles that apply to chemical reactions found in inanimate systems. That is, the amount of energy liberated for comparable degrees of chemical change is the same in both cases. Theoretically when hydrogen combines with oxygen to form water, whether in the living cell or in an inanimate chemical reaction, the same amount of energy is released. In inanimate systems, this energy is liberated as free energy or heat and is lost. In the living cell, although a large portion of the energy liberated from the reaction is lost through dissipation of heat energy, a certain portion, perhaps a third or more, is stored as potential energy for use by the cell. The one significant difference between the animate and the inanimate system is that in cells, the union of hydrogen and oxygen is carried out at the end of a series of oxidation-reduction reactions, which is catalysed by a number of enzymes. At each step a fraction of the total energy of the basic reaction is released, and part of this energy is stored or taken up in the formation of new energy-absorbing compounds as chemical energy. Later as these compounds are broken down upon demand by the cell, the stored energy is released in approximately the same amount as was needed for synthesis of the compound.

It may be helpful at the onset to schematically represent the general outline of cellular oxidations. Taking first the cellular substance or substrate being oxidized, we see that these exogenous or endogenous protein, carbohydrate or fat molecules are first broken down by the removal of two atoms of hydrogen, a reaction catalysed by specific enzymes known as dehydrogenases. These reactions liberate energy which is either dissipated as heat, or is stored as chemical energy in new compounds usually as phosphate esters. As hydrogen is liberated it is carried away by a series of oxidation-reduction reactions, and is ultimately united

with molecular oxygen at the very end of the oxidation or respiratory series. Energy for cell work is stored at each step. Energy is similarly liberated and stored in the process of splitting off of carboxyl groups from certain compounds in the formation of carbon dioxide. Parenthetically it is of interest to note that the bulk of the oxidative systems apparently are found in the large granules (mitochondria) of the cell (4). A schematic diagram of oxidative metabolism (after Stanier (5)).

#### DEHYDROGENASES

The first chemical reaction in the liberation of energy from foodstuffs is the oxidation of the foodstuff or metabolite by the removal of hydrogen. This reaction is brought about by a specific cellular enzyme known as a dehydrogenase. Dehydrogenation can be accomplished by three methods:

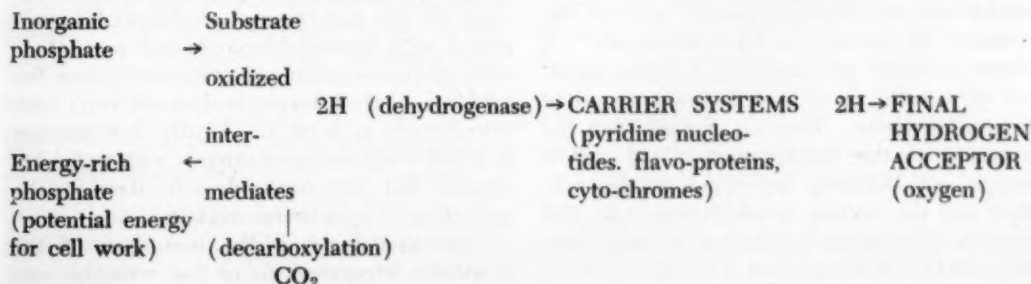
1. The direct transfer of hydrogen from the metabolite to molecular oxygen by a prosthetic group of the dehydrogenase protein so that peroxide is formed. The peroxide is then broken down into water by a catalase or a peroxidase, and the reaction is then complete. D-amino acid oxidase catalysing the dehydrogenation of certain flavoproteins is an example.

2. Hydrogen may be oxidized to water through the mediation of the cytochrome system, between a dehydrogenase system on one hand and molecular oxygen on the other.

3. Hydrogen may be oxidized to water through the mediation of a complex series of oxidation-reduction systems including: dehydrogenase, codehydrogenase (coenzymes, pyridine nucleotides), flavoprotein, cytochrome, cytochrome oxidase, molecular oxygen. Most dehydrogenase systems are of this type.

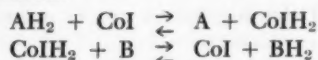
#### COENZYME-LINKED DEHYDROGENASES

In aerobic dehydrogenase systems, the hydrogen liberated from cell metabolites is transferred in one direction toward molecular oxygen, so that the metabolite is oxidized by the removal





of hydrogen and hydrogen is oxidized by union with molecular oxygen. In anaerobic dehydrogenase systems, where oxygen is insufficient to meet the demands of the cell for energy, the hydrogen released may be oxidized by a hydrogen acceptor other than oxygen. A second dehydrogenase system with its own enzyme and substrate is linked to the first system by a specific coenzyme. Hydrogen is transferred and energy is stored without the immediate presence of molecular oxygen. Two coenzymes are known, coenzyme I or diphosphopyridine nucleotide (DPN), and coenzyme II or triphosphopyridine nucleotide (TPN), nicotinic acid amide being an essential component of each. Any coenzyme-linked reaction can be represented as follows:



where A and B are dehydrogenases acting as substrates and CoI is coenzyme I. At the moment of catalysis hydrogen is transferred from the substrate A to the coenzyme I. The reduced coenzyme I must be reoxidized immediately by a new hydrogen acceptor substrate B, in order that the linked dehydrogenase system may continue to react, for there is only a small amount of coenzyme present for reaction. Hydrogen transfer to molecular oxygen must be eventually carried out in order that chemical equilibrium be reached. Hydrogen may be transferred directly to molecular oxygen in a few cases, by way of another hydrogen accepting substrate, or in the case of most coenzyme-linked reactions in the respiratory series, by way of a specific flavoprotein, cytochrome, and finally molecular oxygen. A large number of dehydrogenases are associated with coenzymes, so that one coenzyme may react with a number of specific metabolites in the removal of hydrogen.

#### FLAVOPROTEIN

Flavoproteins are specific riboflavin-containing cellular enzymes that apparently catalyze the oxidation of coenzymes by cytochromes. A variety of these are found in bacteria, yeast, and other cells, in addition to those found in mammalian tissue. They are named, with the exception of the first-discovered "old yellow enzyme" of Warberg, according to the substrate and the manner in which they act. For example, cytochrome *c* reductase in yeast links the reduction of cytochrome *c* by coenzyme II.

Flavoproteins are believed to link the cytochromes with the dehydrogenase systems. Eleven flavoprotein enzymes are listed by Best and Taylor (3).

#### THE CYTOCHROME SYSTEM:

The cytochrome system functions at the extreme end of the respiratory chain of oxidation-reduction reactions, serving to make atmospheric oxygen available to the cell for oxidation of hydrogen which has been liberated from various cellular metabolites by the dehydrogenases at the beginning of the chain. The cytochrome system consists of a group of enzymes related to *heme* found in human hemoglobin. They are widely distributed in nature, being found in plants, certain aerobic bacteria, yeast, and also the muscle of vertebrates. Cytochrome *a*, *b*, *c*, and cytochrome oxidase are the found compounds that make up this system. Their chemical structure is that of a conjugated protein with an attached iron porphyrin or tetrapyrrol prosthetic group, resembling the tetrapyrrol structure of *heme* found in hemoglobin. Their structure also resembles that of chlorophyll found in green plants in which case magnesium replaces the iron atom in the tetrapyrrol structure. It is by means of this prosthetic group, or more specifically the iron atom contained in this prosthetic group, that the reversible oxidation-reduction reactions of cytochromes are possible.

Cytochromes and cytochrome oxidase, although closely related in their iron-porphyrin prosthetic groups are in other ways dissimilar. The cytochrome *oxidase* reacts with small amounts of cyanide to form a completely undissociable compound that inhibits electron transfer and paralyzes cellular respiration. Cytochromes *a*, *b*, and *c* do not react with cyanide. Carbon monoxide forms a similar undissociable compound with cytochrome *oxidase* that paralyzes respiration. In carbon monoxide poisoning, however, tissue poisoning does not occur because the gas first forms a non-dissociable compound with hemoglobin contained in erythrocytes and never reaches the tissues to poison the *oxidases*. Carbon monoxide does not react with cytochromes *a*, *b* or *c*. Finally, cytochromes *a*, *b* and *c* will not react directly with molecular oxygen, but are oxidized only through the mediation of cytochrome *oxidase*.

Fundamentally it is the iron atom of the prosthetic tetrapyrrol group that reversibly un-



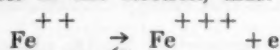
**Cytochromes a, b, or c**

1. Will not react with CN or CO.
2. Will not react directly with molecular oxygen.
3. Molecular weight 13,000.

**Cytochrome oxidase**

1. Reacts with CN or CO to form undissociable compounds that completely inhibit electron transfer and paralyze tissue respiration.
2. Reacts directly with molecular oxygen at pH 7, in the presence of cytochrome.
3. Molecular weight 75,000.

dergoes oxidation or reduction and thus is able to effect electron transfer. Ferrous iron is seen to undergo oxidation to ferric iron with the loss of one electron, thus:



Returning to the path of hydrogen transfer: Phosphopyridine nucleotide (DPN/TPN) transfers hydrogen to a fibroflavine di-nucleotide which in turn transfers hydrogen to cytochrome *b*. Hydrogen ions are furnished to the cytoplasm of the cell, while their electrons are forwarded through the cytochrome system in the order of cytochrome *b*, *c*, *a*, *oxidase*, and finally atmospheric oxygen. The hydrogen unites with oxygen to form hydrogen peroxide which is decomposed by the enzyme, catalase, to water and oxygen. Thus it is that the iron-containing cytochromes, functioning at the very end of the respiratory chain, carry out the final electron transfer of metabolic hydrogen to atmospheric oxygen, and in this way make available to the organism most of the oxygen that it consumes.

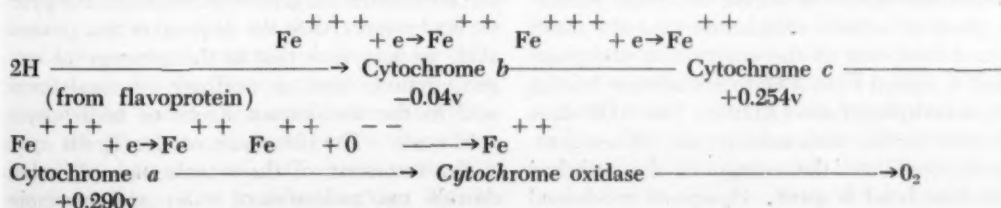
#### THE BIOLOGICAL COUPLING OF OXIDATION AND PHOSPHATE BOND ENERGY

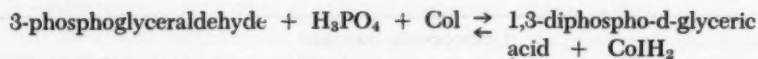
In preceding paragraphs we have seen how cellular oxidations have been effected by the removal of hydrogen from various cellular substrates. It must now be shown how the energy liberated by these dehydrogenations is used by the cell. It must further be shown how this energy is first stored and then released when called for by the cell to do its work.

The highest potential energy for cellular work has been found to be stored chemically in the "high-energy" phosphate bond. Ordinary "low

energy" phosphate ester bonds as typified in glycerophosphate involve a relatively small energy storage of 3,000 calories or less in their synthesis. The "low-energy" of this bond can be greatly increased by a simultaneous oxidation of glyceraldehyde to glyceric acid. Similar oxidative energy-liberating reactions occur in the glycolytic series, the Krebs cycle, and perhaps during the oxidation of lipids. The "high-energy" phosphate linkage represents a chemical compound with larger energy of formation and upon hydrolysis to inorganic phosphate results in the liberation of a quantity of energy comparable with that quantity that went into its formation. For example, at one stage of glycolysis (*vide infra*), inorganic phosphate is taken up by 3-phosphoglyceraldehyde, forming a hypothetical ester, 1,3-diphospho-glyceraldehyde. This compound is dehydrogenated by diphosphopyridine nucleotide (DPN) to 1,3-diphospho-glyceric acid. The change of molecular structure gives the phosphate bond in position *one* a great increase in potential energy. This phosphate radical is now transferred with its high energy content intact to adenosine-diphosphate (ADP), forming the 3rd phosphate group of adenosinetriphosphate (ATP).

Phosphate bond energy has been thus temporarily stored in ATP, ready for release to the cell upon demand, for muscle contractions, intestinal absorption, and other reactions. It has been computed that in the complete oxidation of each unit of glucose from glycogen by way of pyruvate, that 36 high energy phosphate bonds are accumulated and that 345,000 calories of heat energy are produced. Similar coupled





oxidation-phosphorylation reactions with the production of energy-rich phosphate linkages are not confined to mammalian tissues, but are also seen in the conversion of glycogen to glucose in yeast and in certain bacteria. One organism, *Thiobacillus thio-oxidans* is able to store energy-rich phosphate using the energy of sulfur oxidation carried out anaerobically and in the dark (to exclude photosynthesis as a source of energy).

#### CELLULAR RESPIRATION IN MUSCLE

One of the main uses to which the phosphate-bond energy stored in ATP is put is in muscle contraction. Muscle contraction is currently believed to be the shortening of partially unfolded polypeptid chains of actomyosin contained in the micellae of muscle myofibrils, a reaction set off by an electrical impulse or its chemical equivalent entering the muscle from its nerve. Two theories as to the contraction of actomyosin have been postulated. First, "energy-rich" phosphate from ATP passes to actomyosin at the time of stimulation, giving up its energy for work and heat, and leaving ADP plus inorganic phosphate. Second, "energy-rich" phosphate from ATP may pass to actomyosin previous to the muscle contraction, giving up its energy to partially unfold the polypeptid chain to a tensed state, so that it is ready to contract upon stimulation. The one theory regards the relaxed muscle with its extended actomyosin chain as a loose mechanism to be charged only upon stimulation, while the other theory regards the relaxed muscle as a bundle of taut, energy-charged, unfolded actomyosin chains, ready to shorten when nerve stimulation "pulls the trigger."

In either case, the fundamental chemical event in the contraction is the splitting of adenosinetriphosphate (ATP) by an enzyme adenosinetriphosphatase (ATPase), activated by calcium ions before or during the initial anaerobic phase of muscle contraction. As the result of its action, one of the energy-rich phosphate bonds is passed from ATP to actomyosin leaving adenosinediphosphate (ADP). The ADP does not react further with actomyosin. When actomyosin contracts, the energy of the attached phosphate bond is spent. Phosphate is released

as inorganic phosphate which may be reused in the phosphorylation of carbohydrate compounds. High energy phosphate bonds may thus be found in three compounds, actomyosin phosphate, adenosinetriphosphate, and creatine phosphate. The creatine phosphate can be compared to a bank, storing "energy-rich" phosphate bonds instead of money, for use by muscle. Like a bank it needs a teller, in this case ATP, to distribute this energy on demand.

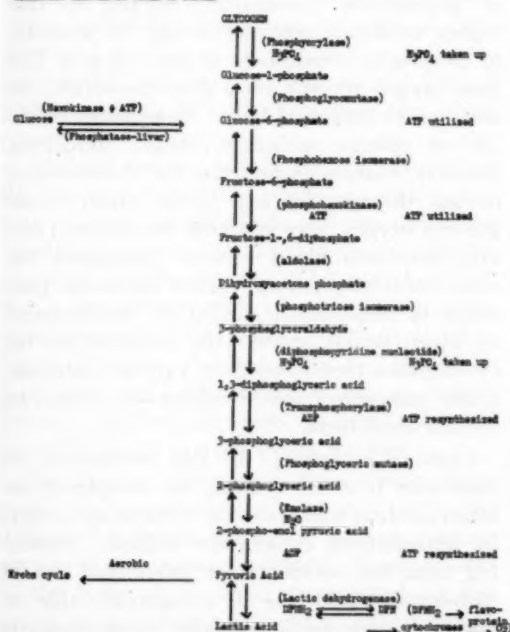
Later in the muscle recovery or aerobic phase, phosphate bonds are generated by glycolysis for transfer to the above compounds. Glycogen catalysed by phosphorylase at the beginning of contraction takes up inorganic phosphate ( $\text{H}_3\text{PO}_4$ ) and simultaneously splits off glucose-1-phosphate. There then begins a series of complex anaerobic transformations of glucose-1-phosphate to its degradation product of pyruvic acid with a production of 29,000 calories of energy. It is at this final stage of glycolysis that we can begin to see some connection between muscle work and more direct oxidation-reduction systems previously discussed. To repeat, under the initial anaerobic phase of muscle contraction, or in periods of sustained contraction and oxygen debt, glycolysis proceeds to the pyruvic acid stage, and this pyruvic acid is eventually converted to carbon dioxide and water with the production of energy and heat. This energy is used for the resynthesis of phosphocreatine and ATP. Pyruvic acid may be reversibly reduced to lactic acid under anaerobic conditions, as in severe exercise. If oxygen then becomes available as it normally would at the conclusion of muscle contraction, the reduced coenzyme ( $\text{DPNH}_2$ ) gives up its hydrogen not to pyruvic acid, but to molecular oxygen through a specific flavoprotein (riboflavin di-nucleotide) and the cytochrome system. As a result, no lactic acid need be formed under aerobic conditions, glycolysis ending at the pyruvic acid stage. As to the disposal of this pyruvic acid, we may note that in the presence of oxygen, pyruvic acid is oxidized via oxaloacetic acid in the well-known Krebs or tricarboxylic acid cycle. The net result of this Krebs cycle is the formation of three molecules of carbon dioxide, two molecules of water, and one mole-

cule of oxaloacetic acid which is reused time and again. In this process, some 325,000 calories are liberated for use chiefly in the resynthesis of glycogen from lactic acid.

The energy made available to the cell for muscular work is summarized in the chart below(3):

Reaction	Purpose	Calories Released
ATP → PC + ADP	Energy for muscle contraction	12,000
CP + ADP → C + ATP	Energy for resynthesis of ATP	10,000
Glycogen to lactic acid (Glycolysis)	Energy for resynthesis of phosphocreatine and ATP	29,000
Oxidation of lactic acid (Tricarboxylic acid cycle)	Energy for resynthesis of glycogen	325,000

#### ANABOLIC GLYCOLYTIC CYCLE OF ENERGY



#### TRICARBOXYLIC ACID CYCLE (KREBS)

In the Krebs cycle a molecule of the 6-carbon compound, tricarboxylic acid or *cis*-aconitic acid is formed by the condensation of one molecule each of pyruvic and oxaloacetic acids, with the release of a molecule of carbon dioxide. *Cis*-aconitic acid is hydrated to form isocitric acid, which is in equilibrium with citric acid, the latter possibly acting as the governor of the over-all speed of reaction of the Krebs cycle. Isocitric acid is oxidized in turn to oxalosuccinic acid. The latter undergoes decarboxylation with the production of the 5-carbon di-carboxylic acid,  $\alpha$ -ketoglutaric acid, with the release of another molecule of carbon dioxide. Further

oxidation results in the formation of the 4-carbon succinic acid and the release of the 3rd and final molecule of carbon dioxide from the original pyruvic acid. Succinic acid is oxidized through fumaric and malic acids to oxaloacetic acid, and the cycle then repeats. At each turn of the cycle, each molecule of pyruvic acid

which enters, yields two molecules of water and three molecules of carbon dioxide, while a molecule of oxaloacetic is reformed. A single molecule of oxaloacetic acid can be used in this way repeatedly in the oxidation of pyruvic acid to carbon dioxide and water; it therefore acts in the manner of a catalyst.

The above reactions producing carbon dioxide require diphosphothiamine as a coenzyme (co-carboxylase) and the various oxidations mentioned are accomplished by specific dehydrogenases, employing phosphopyridine nucleotide or riboflavine nucleotides as coenzymes.

Pyruvic acid is in equilibrium with members of the tricarboxylic acid cycle. This cycle is probably the final common path for much of the carbohydrate, fat and protein metabolism. Its reactions liberate energy, and most of this energy is used for the establishment of energy-rich phosphate bonds.

#### CARBON DIOXIDE FIXATION

Not all carbon dioxide liberated in the course of catabolism is excreted, but a certain portion is utilized by the tissues for the synthesis of complex carbon containing compounds such as glycogen. Carbon dioxide utilization or *fixation* is possible by two known mechanisms. First of all Werkman and Wood(6) have postulated that glycogen can be formed by the condensation of pyruvic acid and carbon dioxide. Hastings et al(7), using carbon dioxide containing radioactive carbon ( $C^{14}$ ) have shown in rats that carbon dioxide is incorporated into liver glycogen laid down when lactic acid is fed, presumably from pyruvic acid condensation with carbon dioxide. Carbon dioxide fixation also occurs in the isocitric acid,  $\alpha$ -keto-glutaric acid and succinic acid reactions of the Krebs cycle.





chromes, and saving the patient.

*Diphtheria toxin* has recently been thought to in some way immobilize cytochrome *b*, and thus exert its toxic effect.

*Cytochromes intravenously.* It might be well to ask why supplemental cytochromes could not be given in instances where poor tissue oxygenation might be expected. Cytochrome *c* is the only cytochrome that has been obtained in pure form so far, and it has been given to subjects in which artificially induced anoxia produced abnormal EKG tracings. 50-60 mgm of cytochrome *c* given intravenously was found to revert these EKG tracings to normal, although the authors(8) doubted the ability of the enzyme to enhance the myocardial utilization of oxygen. Other authors(9) found that 50 mgm of cytochrome *c* given intravenously had no ability to increase the capacity for effort without pain of patients with angina of effort over that effect produced by placebos.

*Alkaloids and Anaesthetic Agents.* Turning to the opposite end of the respiratory chain, it is now believed that alkaloids and probably most anaesthetic agents have their depressant action on tissue dehydrogenases. This would suggest that in the administration of certain anaesthetics, while one end of the respiratory chain is selectively depressed, that the other end should be fully activated. The generous use of oxygen during anaesthesia in order to fully oxygenate the cytochrome system seems justified.

*The Role of Vitamins in Intracellular oxidations.* Certain vitamins are essential to the formation of hydrogen carriers in the intermediate portion of the respiratory chain. Nicotinic acid in the form of its amide is required for the formation of coenzyme I and II (the pyridine nucleotides). Riboflavin is required for the formation of (ribo) flavine nucleotides. Pyridoxine (B-6) has recently been discovered to be essential in transamination, in which amino groups are transferred from one compound to another. Pantothenic acid is known to be involved in certain acetylations, and vitamin B-12 has been discovered to speed the conversion of purine in nucleoprotein to uric acid by xanthine oxidase. A good dietary regimen, adequate in these vitamin factors probably insures the proper function of these phases of respiration, especially in times of stress, such as in the acute depression of one part of the respiratory chain by an anaesthetic or toxic agent. Vitamin

B-1 in the form of diphosphothiamine is an essential part of the coenzyme, cocarboxylase, which makes possible the catalysis of carboxylic acid to carbon dioxide. This reaction is the only important source of carbon dioxide of metabolic origin. Cocarboxylase also helps in the resynthesis of glycogen from carbon dioxide, and is thus necessary for carbon dioxide fixation.

*The Role of Hormones in Intracellular Respirations.* It is now felt that all hormones have some effect in influencing the rate of certain of the respiratory reactions. From work done by Cori et al (10 and 11) it was discovered that hexokinase in the liver catalysed the phosphorylation of glucose by ATP to glucose-6-phosphate and that this reaction was inhibited by the diabetogenic hormone of the anterior pituitary, reinforced by a hormone from the adrenal cortex. Insulin was found to remove this inhibition. The question of the effect of thyroid hormone on the exchange of liver ATP in these phosphorylations has not been settled by radioactive isotope studies(12).

At least one of the adrenal cortical steroids, desoxycorticosterone, has been found to inhibit D-amino acid oxidase(22).

Undoubtedly many drugs have their action dependent on alteration of the respiratory enzymes or their rates of reaction. Only one of these will be mentioned because of its recent clinical interest. Tetraethylthiuran disulfide ("Antabuse") is a compound without pharmacological action in itself. When alcohol is taken following antabuse ingestion, there occurs an uncomfortable clinical reaction consisting of nausea, vomiting, generalized vasodilatation, correlated with an increase in blood acetaldehyde. These same symptoms can be reproduced by the administration of acetaldehyde. It has been postulated that antabuse inhibits xanthine oxidase, one of the cellular dehydrogenase systems capable of oxidizing acetaldehyde, thus causing an accumulation of acetaldehyde in the blood(13).

#### SUMMARY

Cellular respiration has been discussed from the point of view that it consists of all those biochemical reactions, usually oxidations, that convert bound energy in foodstuffs into free energy for cell use. The manner in which these oxidations have been effected through the removal of hydrogen and carbon dioxide has been detailed, and methods by which the cell stores

this free energy at higher energy potentials has been indicated. Finally certain clinical aspects of the physiology of cellular respiration have been briefly mentioned.

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## PHOENIX *Clinical* CLUB

### MASSACHUSETTS GENERAL HOSPITAL CASE NO. 29462

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the *New England Journal of Medicine*. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

An eighteen-year-old boy was first seen in the Out Patient Department, about one year before the present admission because of hemoptysis of four years' duration.

At the age of thirteen, while playing, he coughed up several large clots of blood. He had a similar episode one week later, and there were repeated intermittently up to the day of his visit to the Out Patient Department. The amounts raised varied from a speck to half a cupful. No definite statement was made concerning the frequency of attacks, but "at no time were they more than two weeks apart." There were no chills, fever, night sweats or chest pain until "recently," when he had noted sharp twinges of pain in the right upper chest unassociated with cough or hemoptysis. There had been no history of inhalation of foreign body, antecedent upper respiratory infection, known contact with tuberculosis or undue bleeding tendency. He had fractured the left collar bone one year before onset of his symptoms, he was in a cast for two months.

Examination in the Out Patient Department showed a well-developed and well-nourished boy in no distress. The chest was well developed. There was dullness over the right upper lobe posteriorly to the midscapular region and anteriorly to the fourth rib, with occasional expiratory bubbling and coarse crepitant rales posteriorly. Kronig's isthmus was slightly narrowed on the right. The lungs were otherwise clear. The heart was in its usual position and normal in size. The abdomen was negative. There was no clubbing of the fingers.

The blood pressure was 138 systolic, 68 diastolic. The temperature was 98°F., the pulse 60, and the respirations 20.

X-ray examination of the chest showed a round, homogeneous soft-tissue mass in the upper posterior mediastinum measuring approximately 11.5 cm. in diameter, without any evidence of calcification or cavity formation. The tumor obliterated the upper portion of the right lung but the visible portion of the right lung and the left lung appeared clear. The hilar shadows were normal and equal on both sides. The heart was not remarkable. No definite bone involvement could be seen, but there was a questionable pressure defect on the right lateral aspect of the fourth dorsal vertebra, with narrowing of the right pedicle. The trachea appeared straight in the midline and there was no mediastinal displacement. No fluid was present. Bucky films of the chest confirmed the

presence of the large mass in the lung. An area of probable slight erosion of the upper aspect of the sixth rib was also seen. There was an irregular air pattern in the region of the mass, probably due to partially collapsed lung that was superimposed on the shadow of the mass.

While being followed in the Out Patient Department, the patient developed wheezing, especially at night. No other information was given regarding these attacks except that "he was quite free of wheezes at times." He was free of pain and maintained his weight. Bronchoscopy showed "extrinsic pressure but failed to reveal the source of the bleeding, although a moderate amount of bright-red blood was seen in the trachea and right main bronchus." Hospitalization was advised but refused.

For the next ten months he remained free from pain, hemoptysis and cough. At the end of this period he obtained a job in a weaving mill, doing heavy labor. Eight days before entry he suddenly became dyspneic while working. The dyspnea forced him to leave his work. Six days prior to admission he developed severe pain in the right anterior chest and he was admitted to a community hospital, where x-ray films showed a lobulated tumor mass in the upper half of the right lung, with pleural effusion. Two chest taps yielded 100 and 300 c.c. of reddish-brown pleural fluid and gave considerable relief. He was transferred to this hospital.

Physical examination showed a poorly nourished, cyanotic and dyspneic man with anxious, pale facies and visible flaring of the alae nasi on breathing. The right chest was splinted during inspiration. There were dullness and diminished tactile fremitus and breath sounds over the right upper and lower lobes. Dullness was obtained over the right middle lobe, with egophony and increased tactile fremitus. Pectoriloquy was heard over the right upper lobe posteriorly and over the middle lobe anteriorly. The diaphragmatic excursion was poor on the right. The heart was normal. The remainder of the examination was not remarkable.

The blood pressure was 130 systolic, 60 diastolic. The temperature was 99.6°F., the pulse 128, and the respirations 25.

Examination of the blood showed a red-cell count of 4,100,000, with 75 per cent hemoglobin. The white-cell count was 11,000 with 89 per

cent neutrophils. A blood Hinton test was negative. The prothrombin time was 28 seconds (normal 16 seconds); the hematocrit was 39.2 per cent. The nonprotein nitrogen was 10 mg. per 100 c.c., and the protein 5 gm. The van den Bergh test was normal. Urinalysis was negative.

X-ray examination of the chest, almost a year after the first one, showed the tumor mass still present. The right pleural cavity was filled with a fairly large amount of fluid and there was partial collapse of the right middle and lower lobes, which had a honeycombed appearance. The left lung appeared clear, but there was questionable crowding of the markings in the left lower lobe.

During the next ten days, three chest taps yielded 2800, 1500 and 1000 c.c. of dark bloody fluid, which on smear and culture was negative for organisms. No tumor cells were seen in the sediment.

Repeated x-ray studies after the taps showed that, instead of a fluid level, there was an area of increased density with irregular markings extending along the lateral axillary line. There were numerous irregular areas of decreased density in the right upper chest, either in the tumor or in the pleural cavity. None of the visualized bones, especially the ribs, showed evidence of destruction and no other changes were noted. Another bronchoscopy, year after the first, was negative. No evidence of intrinsic or extrinsic tumor was seen.

On the eleventh hospital day an operation was performed.

DR. LOUIS B. BALDWIN:

The outstanding features of this case are the long duration of the illness with frequent hemoptysis but without other symptoms until about five years after the onset, and the period of ten months when there was no chest pain, no hemoptysis and no cough. Following this period however there was the acute advent of marked dyspnoea and the rapid reaccumulation of bloody pleural fluids.

When first seen in the out-patient department he had been coughing up blood frequently for four years. He was in good general physical condition, but the physical signs pointed to a lesion in the right upper chest. A detailed x-ray report, at this time, indicated that there was a homogeneous tissue mass in the upper posterior mediastinum, without evidence of calci-



fication or cavity formation. The hilar shadows were normal and equal on both sides. Bronchoscopy showed extrinsic pressure but failed to reveal the source of the bleeding.

In spite of the thorough roentgenological studies we are still in some doubt at this point whether we are dealing with a tumor originating in the mediastinum, in the pleura, or in the lung, or in the bony framework of the chest. It would be hard to conceive of a tumor in the posterior mediastinum producing early hemoptysis and it would be equally difficult to picture a tumor of the pleura with invasion of the lung, remaining silent except for moderate chest pain and hemoptysis for so long a time. If we can put any reliance on the protocol it is inconceivable that we are dealing with a bronchogenic carcinoma, not only because of the age of the patient, and the description of the mass but also because of its subsequent course.

A tumor originating in the bony framework such as osteosarcoma can be readily ruled out by the repeated x-ray studies which revealed no bony involvement. Primary neoplasm of the pleura must be seriously considered in spite of the fact that they are rare. They may be benign or malignant originating in either the subserous layer or the surface of the pleura. These tumors may be localized or diffuse. Golden states that localized pleural neoplasms may exist as pedunculated masses and are generally fibrosarcomas of low malignancy.

By x-ray they appear sharply demarcated, rounded or oval, and may or may not show a lobulated border. They are very difficult to differentiate from a primary carcinoma of the lung. They grow slowly, sometimes existing for many years. Eventually, unless they metastasize they fill the pleural cavity and cause death by interfering with pulmonary circulation.

So call endotheliomas or mesotheliomas have their origin in the surface lining of the pleura. They may also appear by x-ray as a rounded, sharply demarcated mass. These tumors may be slow growing and tend to infiltrate the outer zone of the lung, dipping into interlobar fissures. As a rule, carcinomas associated with massive pleural involvement metastasize late in the disease. Rubin states that sooner or later neoplasms of the pleura usually cause massive sero-hemorrhagic effusions which rapidly re-accumulate in spite of repeated chest aspirations.

If this patient has a tumor of the pleura it cannot be the non-invasive benign tumor such as fibroma, lipoma, and chondroma.

How does the consideration of a pleural mesothelioma fit this case?

To be sure the duration of five years or longer makes the diagnosis questionable; for these tumors rarely last more than two years before invading the pericardium and the diaphragmatic and mediastinal reflections of the pleura. They may extend to involve other organs and tissues. It is also difficult to conceive of a mesothelioma causing hemoptysis for four years or longer without other symptoms.

The same objection may be made in the consideration of a fibrosarcoma of the pleura, moreover these tumors rarely, if ever, invade the lung to produce hemoptysis.

But whatever the primary condition characterized by an upper right chest mass and hemoptysis the later developments and x-ray studies indicate extensive pleural involvement with a bloody pleural fluid and an area of increased density with irregular markings extending along the lateral axillary line. It is to be noted that malignant neoplasms of the lung and pleura are the cause of from 65 to 85 percent of hemorrhagic pleural effusions. In Berliner's material consisting of 120 cases, tumor cells were found in 50% of the hemorrhagic fluids complicating malignant neoplasms. The failure to find tumor cells in the present instance therefore does not prove that malignancy is ruled out.

Hemorrhagic pleural effusions are common in pulmonary infarction and infrequent in tuberculosis. They may be due to local circulatory disturbances, such as pressure exerted by an aortic aneurism or tumor on a large intrathoracic vein. Tumors in the anterior mediastinum are more likely to produce this condition, such as thymic tumors or teratoid tumors. Harrington cites an instance of a benign tumor associated with a bloody effusion.

Next in importance diagnostically are the mediastinal tumors. The lymphomas such as Hodgkins disease and lymphosarcoma are often first detected in the mediastinal lymph nodes. The former is more common in youth and roentgenologically is characterized by enlarged discrete hilar nodes extending laterally from the mediastinum, usually bilaterally. They are either sharply circumscribed or lobulated or fus-



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(The Council on Pharmacy and Chemistry of the American Medical Association has adopted the following statement of Actions and Uses and of Dosage for publication in connection with a description of Banthine Bromide for inclusion in New and Nonofficial Remedies)

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Clinical studies indicate that the drug effectively inhibits motility of the gastrointestinal and genitourinary tracts and, to a variable degree, diminishes the volume of perspiration and salivary, gastric and pancreatic secretions. It also decreases mucoprotein secretion. Like atropine, it produces mydriasis and cycloplegia when applied locally to the eye or administered systemically, but until more clinical evidence becomes available, its local use for this purpose is not recommended. The value of the drug for preventing abnormal cardiac reflexes through the vagus during thoracic surgery, or as an agent for routine preoperative medication in place of atropine, requires further investigation before final conclusions can be reached.

Methantheline bromide is indicated for clinical use whenever anticholinergic spasmolytic action is desired, provided it is not contraindicated because of its atropine-like characteristics or because of a patient's intolerance to the unavoidable side effects of such therapy. It is useful as an adjunct in the management of peptic ulcer, chronic hypertrophic gastritis, certain less specific forms of gastritis, pylorospasm, hyperemesis gravidarum, biliary dyskinesia, acute and chronic pancreatitis, hypermotility of the small intestine not associated with organic change, ileostomies, spastic colon (mucous colitis, irritable bowel), diverticulitis, ureteral and urinary bladder spasm, hyperhidrosis or control of normal sweating which aggravates certain dermatoses, and control of salivation.

Methantheline bromide produces some degree of cycloplegia and mydriasis in therapeutic doses and

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**Dosage.**—Methantheline bromide is administered orally or parenterally by either the intramuscular or intravenous route. Parenteral administration is not advised for patients able to take the drug orally. The average initial adult dose, oral or parenteral, is 50 mg. For patients with considerable intolerance, 25 mg. may be employed. In the management of peptic ulcer, a beginning schedule of 50 mg. three times daily before meals and 100 to 150 mg. on retiring is suggested. However, the usual effective dose is 100 mg. four times daily, although some patients may require more or less than this amount. The dosage may be increased to tolerance, using dryness of the mouth as a guide, and adjusted to meet the individual response of patients. Maintenance dosage in peptic ulcer is usually considered to be about one-half the therapeutic level. In the management of other hypermotile or hypersecretory states, the dosage should be adjusted to the smallest amount which will relieve the symptoms. When spastic conditions are secondary to inflammatory or other organic lesions, therapy directed toward the cause should be employed whenever possible.

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Tablets Banthine Bromide: 50 mg.

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ed into large masses. A delicate reticulation is often seen extending into the adjacent lung tissue. There is seldom intrathoracic displacement. There may be widening of the right mediastinal shadow extending to the root of the neck but unlike the description in this case the borders are straight. The lungs are involved in 13 percent and the pleura in 11 percent in 214 cases studied by Pierce, Jacox, and Hildreth. Moulten found pleural involvement in 9 of 18 cases of Hodgkins. The fluid may be serous, chylous, or even bloody. The majority of the pleural lesions consist of scattered nodules or flat infiltrations. But extensive indurations involving also the ribs and spine are not uncommon. Radiotherapy is effective even if the pleura contains considerable fluid. When the lung is involved there may be hemoptysis.

Surely Hodgkins disease or lymphosarcoma in this case could have been easily ruled out by the use of radio-therapy. Mediastinitis and sarcoidosis may be mentioned in passing but are obviously not involved in this case.

Of the mediastinal tumors there are those of the anterior and posterior mediastinum. Among the former are the teratoid tumors which are usually cystic structures. They often contain all types of tissues, not only undifferentiated cellular elements, but also rudimentary organs. Solid tumors are less frequent and more liable to undergo malignant changes. The fact that in the present instance the mass is described as being in the posterior mediastinum rules out this diagnosis. For the same reason thymic tumors and intrathoracic thyroid are ruled out.

In the posterior mediastinum are found the neurogenic tumors. They arise from the spinal, intercostal, and sympathetic nerves and ganglia. Kent and his co-workers found only two instances of neurogenic tumors in the anterior mediastinum. These tumors have been found accidentally in the course of routine roentgen examinations. If symptoms are present, their nature depends on the size of the tumor, the nerves involved, the presence of malignant changes. Pressure may produce a Horner's syndrome or hoarseness. By roentgenology areas of calcification may be seen. A diagnostic pneumothorax reveals the tumor to be extra pulmonary. Lobulation of the tumor is suggestive of malignancy. Erosion of ribs or vertebral bodies may be caused by pressure alone.

It is difficult to conceive of a neurogenic tumor

causing hemoptysis for five years without there being metastatic lung involvement. It is however possible that enough pressure was exerted by the growth on the lung to produce changes in the lung or smaller bronchial tubes, or there might have been actual invasion of the lung by the growth. Tumors such as bronchogenic cysts and cystic hygromas do not produce the clinical picture seen in our case. There are other rare tumors however which may occur in the posterior mediastinum such as fibrosarcomas or myxosarcomas.

It is obvious that our patient does not have a bronchogenic carcinoma. His age and the course of the illness are against this diagnosis. But he might have a bronchial adenoma. These tumors arise in the bronchial mucus glands or in their ducts. They are either globular or pedunculated. They often originate within the bronchial wall and extend into the peribronchial tissue. A few instances have been reported with regional and even distant metastases. Adenomas usually occur in young individuals. The tumor chiefly affects females and predominantly the left lung. The outstanding symptom is recurring hemoptysis. The physical and roentgen findings are not unlike those encountered in bronchogenic carcinoma. These tumors usually remain within the lung but rarely they may extend into the mediastinum. I do not feel that this patient had a bronchial adenoma.

In conclusion we have an 18 year old boy who for years has had a mass in the upper posterior mediastinum without evidence of lung involvement, except for a few rales heard at the right apex and attacks of wheezing, which might be explained by pressure of the mass on the lung. There were no constant changes noted in the ribs and vertebrae in serial studies, but there was a prolonged history of hemoptysis, and the sudden final breakdown occurred after a period of heavy work. The later x-rays showed the same mass presumably unchanged in size but with an area of increased density along the lateral axillary line. This is interpreted as showing evidence of pleural metastasis but the meaning of the other findings is not clear, unless the areas of decreased density in either the pleural cavity or the tumor are further evidences of malignant changes. We have no evidence of a distant metastatic focus such as a testicular teratoma or a renal hypernephroma.

My first choice in diagnosis is, Mesothelioma of pleura; (2) Neurogenic tumor of mediastinum; (3) Bronchial adenoma (very unlikely)

#### DIFFERENTIAL DIAGNOSIS

Dr. Austin Brues: I should like to discuss this case mainly from the standpoint of its natural history, which seems rather specific and characteristic. The boy had a lesion that had apparently existed for at least five years and first gave symptoms at the age of thirteen. The history divides itself in to three phases. First, there was a long period in which the only symptom was rather persistent hemoptysis. Secondly, after four or more years there was a period of wheezing, suggesting bronchial compression. Finally, there was an acute sudden episode with pain in the anterior chest and dyspnea, and the rapid development of bloody pleural effusion. To this history we can add the finding of a round mass inside the chest, apparently arising from the posterior mediastinum, and presumably the seat of the symptoms. We have no evidence of disease elsewhere in the body, except that he had a prolonged prothrombin time and a low serum protein, however, the van den Bergh test was normal, and we have insufficient evidence to implicate the liver.

The question that we have to settle is, What sort of isolated mass in this location will progress to give this sort of history? Obviously this was not an acute infectious process. It might for hemoptysis, and there was no evidence of the bronchial nodes or to tuberculosis arising from the spine. The entire early course of the process, however, was essentially silent except for hemoptysis, and there was no evidence of infection or tuberculosis elsewhere. I consider that an infectious process of any sort is unlikely. A hydatid cyst of the mediastinum, first giving rupture into the bronchus, later into the pleural cavity, might give rise to this sequence of symptoms; but we have no reason to suspect an unusual environmental association and the patient had no eosinophilia. Moreover, no eosinophilia was noted even after extension of the process into the pleural cavity, which, if due to rupture of a hydatid cyst, would have caused a certain amount of the cyst contents to be absorbed.

In a young patient we must think of a congenital anomaly-bronchostenosis or a lung cyst. A condition of this sort would most likely

progress to serious symptoms only if it became secondarily infected. Again I point out that infection was not a perceptible element in the clinical picture.

This brings me to tumor. The long duration is in favor of a benign tumor or a tumor that underwent malignant change late, that is, during the last year. Lymphoma can exist for a period of many years without characteristic progression. Such a lymphoma would be the giant follicular type or a very scirrhous type of Hodgkin's disease. I should point out that hemoptysis is a rare symptom in lymphoma, and if it does occur it suggests that one is dealing with a particularly invasive form, which would not be the type giving a long, relatively quiescent history. It seems to me the presumptive diagnosis is a teratoma, not well enough differentiated, as is the classic dermoid cyst, to show calcification by x-ray or to yield hairs or other recognizable elements in the sputum or pleural fluid. The life history of this process is emphatically in favor of this diagnosis. These tumors appear early in life, may give their first symptoms at puberty or later, and sometimes give hemoptysis over a long period. Eventually they are likely to undergo malignant change or to rupture. I should be in favor of rupture as the explanation of the final episode here because of the extreme acuteness of its onset, although invasion of the pleura is a possible explanation, and would point to malignant change. I do not believe that it is necessary to assume, however, that the process had become malignant.

The fly in the ointment is the location of the lesion. It was apparently posterior in position, whereas dermoid cysts and teratoid tumors are almost always found in the anterior mediastinum.

Typical neoplasms of the posterior mediastinum are of neural origin. The history, however, is against one of these. They grow behind the pleura, and one would not expect cough and hemoptysis to be the earliest symptoms. Because of their location they frequently produce Horner's syndrome or pain early in the course of their development. The first symptom here was hemoptysis, which suggests that the lesion was near the bronchi and not posterior to the pleura. There are various rare tumors, such as chondroma, endothelioma, angioma and lipoma, that might arise in this location. There is one

thing somewhat in favor of the possibility of an angiomatous tumor—the characteristic picture of bleeding with which the history started.

I wonder if we might see the x-ray films.

Dr. Laurence L. Robbins: These films show a fairly smooth mass arising posteriorly in the mediastinum, or apparently arising in the mediastinum. The one thing in favor of the neural type of tumor, in addition to its location posteriorly, is that there is a definite erosion of the pedicle of the fourth thoracic vertebra. So far as the ribs are concerned, I am not at all sure that there are any definite changes. This is the shadow described as an irregular area overlying the mass and representing aerated lung. I am not convinced of that. The only other possibility is that it is fat within the tumor. It is difficult to be sure, and most likely it is air in the overlying lung.

These are the films taken at the time of the second admission; they show a large amount of fluid in the pleural cavity and disease in the right lower lobe. The mass and the shadow, which is either air or fat, are still present.

This view, with the patient on his side, was taken after a tap to determine whether there were pleural metastases. There are irregularities along the pleura, but they are most likely masses of fibrin rather than metastases, because fibrin is usually present with bloody fluid. One cannot, however, positively rule out metastases.

Dr. Brues: I am going to let my diagnosis rest on the history rather than on the location and to assume that the tumor arose near the bronchi rather than posterior to the pleura. I shall say that it was a teratoid tumor.

Dr. Richard H. Sweet: Might I say as a representative of the service that saw the man before he was operated on that we did not believe, at least I did not believe that he had a benign tumor. In fact I have never seen a benign mediastinal tumor produce bloody effusion. My personal diagnosis was, and I suspect some of the others who saw the patient were of the same opinion, a malignant tumor, probably teratoma. Dr. Brown operated on this patient. What does he say?

Dr. Robert K. Brown: We did a thoracoscopy and did not see much except a large empty space with implants of fibrin on the chest wall. Biopsies were taken of these and they were not malignant. Since we still thought that the tumor was malignant, the patient was explored.

The preoperative diagnosis was malignant neurogenic tumor or neurofibrosarcoma, because we thought that it arose in the posterior mediastinum.

At operation there was a large cystic mass in the upper part of the right thorax. This contained a hole, and the rupture was probably the cause of the bloody pleural fluid. A biopsy specimen was taken from the wall of the cystic mass at the edge of the rupture, and on cutting, it was evident that there was lung tissue adherent to the wall of the mass. I thought this was collapsed lung plastered over the cyst. The patient died following operation.

Dr. Benjamin Castleman: Would anyone want to venture a diagnosis at this point?

Dr. Ralph Adams: I have no idea what was wrong with this man. I think that the lesion arose in his lung because of two factors that almost have to place it in the lung. First, there was frequently recurring hemoptysis. There was a rule here a few years ago that daily hemoptysis meant carcinoma. After three years we found an exception in a case of bronchiectasis. That was the first exception, and I have since encountered one other. In the second place, this man had a wheeze, indicative of developing bronchial obstruction. My personal diagnosis, if I were about to operate, would be slowly growing sarcoma of the lung. I believe that sarcoma in the lung has occurred in this hospital only once.

Dr. Sweet: That is correct.

Dr. Robbins: How do you explain the erosion of the pedicle of the fourth thoracic vertebra if it is a primary lung tumor?

Dr. Adams: I could not explain it as a primary lung tumor unless it were sarcoma of the lung. If it were a sarcoma arising in the mediastinum and invading the lung the explanation would be easy.

Dr. George W. Holmes: Is there anything to show why there was such a delay between the first roentgenologic examination and operation.

Dr. Castleman: The patient refused operation.

#### CLINICAL DIAGNOSIS

Superior mediastinal tumor, with rupture into the pleural cavity.

#### DR. BRUES'S DIAGNOSIS

Teratoid tumor of mediastinum, with rupture into the pleural cavity.



### ANATOMICAL DIAGNOSIS

Neurofibroma of lung, with cystic degeneration and rupture into the pleural cavity.

### PATHOLOGICAL DISCUSSION

Dr. Castleman: The material that Dr. Brown removed from the cyst or tumor was composed of spindle-shaped cells, some in whorls, which strongly suggested neurogenic benign tumor. In favor of this was the presence of huge numbers of so-called "foam cells," that is, large lipoidladen mononuclear cells. That is probably the reason why Dr. Robbins thought he saw fat in the tumor. Certainly a large part of the tumor was composed of cells filled with fat. Lipoid degeneration of a neurofibroma is not at all uncommon. We were fairly certain that the tumor was benign.

The autopsy specimen shows that this tumor was within the lung. It had broken down so much that it had become cystic. This cystic mass, which was about 7 or 8 cm. in diameter, communicated with the apical branch of the right upper lobe bronchus, and this certainly

accounted for the long-standing hemoptysis. I believe that the tumor probably arose close to the bronchus, or perhaps in it, and degenerated slowly as it gradually increased in size. There was lung tissue surrounding the cyst on all sides except over the area where it had ruptured.

We have had 2 other cases of neurofibroma of the lung. One was in a man of thirty who had all the signs and symptoms of a bronchial adenoma, that is, bronchial obstruction with hemoptysis. Bronchoscopic biopsy showed it to be a neurofibroma, and Dr. Benedict removed the tumor via the bronchoscope in three operations.

Dr. Edward B. Benedict: The patient was completely relieved by bronchoscopy. I have not seen him for three years. He had no apparent lung damage.

Dr. Castleman: The other case was one in which Dr. Churchill performed a lobectomy for a well-circumscribed tumor that pressed on a bronchus but had not actually invaded its mucosa.

## BOOK REVIEW

*New and Nonofficial remedies.* Issued under the direction and supervision of The Council on Pharmacy and Chemistry and American Medical Association. J. B. Lippincott Company Philadelphia 1951.

This volume is too well known to most physicians to require extensive comment. It is pleasant to note that the authors are apparently on the alert for means of improving the book as each edition seems to show considerable improvement over the previous one. In the preface the authors invite criticism with a view to further improvements of the book. The book is really divided into four parts, the first part giving the names of the members of The Council on Pharmacy and Chemistry with their consultants and outlining the purposes of The Council on Pharmacy and Chemistry and giving the requirements for admission of articles to this volume. Other information such as general provisions and labeling requirements is also found here.

The next section is called section "A" and entitled Accepted Products. Here the products are listed according to their place in 23 classes

of drugs or therapeutic agents. As an example of the type of classification used the drugs are listed under such classifications as analgesics, anesthetics, antispasmodics, autonomic drugs, contraceptives, diuretics, etc. Here are the essential therapeutic information about the drugs is found and the preparations made by various manufacturers are listed.

In section "B" the tests and standards for non-official products are given. I think this was an improvement to place this information in a separate section since it is readily available here and does not get in the way when one wishes to read up on a drug for therapeutic purposes only.

Section "C" contains a comprehensive bibliography with reference to the products listed. There is also an index to distributors of the preparations listed as well as a very good general index.

This is valuable up to date reference material. I think every physician who has this volume available will find himself referring to it frequently.

# THE *Secretary's* MESSAGE

## NATIONAL ADVISORY COMMITTEE TO SELECTIVE SERVICE SYSTEM

Bulletin Volume II, No. 7

### *Release from Service*

Navy Policy regarding physicians and dentists (former registrants and reservists):

Priority I .....	24 months
Priority II .....	12-24 months
Priority III .....	24 months
Priority IV	
(12 months during World War II) .....	12-17 months
(Less than 12 months during World War II) .....	12-24 months

### Army policy:

Any physician or dentist who served 12 months during World War II becomes eligible for release after 17 months of current service, except those called in Organized Reserve Units, who will serve the full 24 months.

### Air Force policy:

Any physician, dentist or veterinarian may request release after 21 months of service. If he was in Priority I or II the 21 months must be during the present term of service.

### *Induction Group*

Requests for delay in active duty orders, asked for some of those physicians who accepted commissions after receiving orders for induction and before being inducted, have been determined "entirely out of order" except under very special extenuating circumstances. Health Resources Advisory Committee is calling all records of such cases to Washington for review.

### *Physician Draft*

Planned induction under Selective Service of 333 Priority I medical officers scheduled during the month of August has been delayed temporarily at request of Department of Defense. This delay is based entirely upon an unanticipated and sustained increase in number of doctors applying for Reserve Commissions in the Armed Forces. Whether doctors whose induction was delayed during August will be called during September is dependent upon volunteer rate during next few weeks. No reduction anticipated for call of 152 Priority I doctors scheduled to be inducted in September.

### *Physician Inductions*

Many special registrants ordered by their local boards to report for induction on the July call for 717 doctors were found to have been previously commissioned. Since the July call, the Armed Forces have commissioned 429 doctors. Of this number, 333 received Army commissions, 186 Air Force, 3 Navy, and 7 were commissioned by the United States Public Health Service. Incomplete state reports disclosed that 10 doctors were declared physically unfit; 80 were reclassified, postponed, or cancelled; 2 were inducted; an indefinite number are still pending for one reason or another; and another very small number are believed to be delinquent and will be reported as such by their local boards. Of the two inducted, it has recently been reported in the public press that one of them agreed to accept a commission after completion of 3 days' service as an enlisted man.

# Editorial

## ARIZONA MEDICINE

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The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION. (See MEDICAL WRITING by Morris Fishbein.)
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Submit manuscript typewritten and double-spaced.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.

The Editor is always ready, willing, and happy to help in any way possible.

### CAREFUL, DOCTOR!

In recent years the number of organizations who are interested in the medical patient's welfare has increased tremendously. Besides the insurance companies who have long been with us we now have such organizations as American Cancer Society, Foundation for Infantile Paralysis, American Heart Association, and numerous other welfare agencies from the national level

on down to the local levels. There are various disease registries such as the Tumor registry and the cancer registries in various states.

Any physician may receive requests from any or all of these agencies for information about his patients and these requests may become so numerous and so routine that their validity may be taken as a matter of course. The physician is then on dangerous ground as he is dealing in privileged communications under the law and should he neglect to obtain a release from the patient before he supplies the information requested he is leaving himself open to legal action.

Many times the person or organization requesting the information, because their interest is valid and humanitarian, apparently feel that they are exempt from this rule. A single illustration will demonstrate this. During the last war I had a patient who was dying of cancer. She had a son on duty overseas in the armed forces. The Red Cross was trying to obtain a leave of absence for the boy to visit his mother. I knew nothing of this until I received a call from the Red Cross asking for information about the patient. The young lady who called was quite surprised when I insisted on a release from the patient and replied to my insistence by informing me somewhat haughtily "Why, this is the Red Cross." I patiently explained to her that no matter how high her motive, how worthy the cause, or how poignant the situation, that nevertheless the fact remained that I was dealing in a privileged communication and would need the patient's permission for its release.

There are very few occasions when you can legally reveal information about your patient without his consent. Even on the witness stand in court you are not required to do so unless ordered to by the judge. Ordinarily you may answer safely in court as the failure of your patient's counsel to object to a question implies his consent for you to answer it. If there is any doubt however in your mind, indicate that you consider this something you cannot answer without consent of the patient and ask the judge for a directive. If he orders you to answer you are legally absolved of the consequence.

The growing desire of the medical profession for better public relations and particularly as it concerns better cooperation with the newspapers in news of prominent individuals can be a trap and a snare. The press has long contended that doctors are too close-mouthed and "standoffish" and will not supply the news which "the public deserves". The physician

caring for a prominent patient, anxious to offset this feeling of the press may easily be led to divulge something as news to which the patient may object.

So! Careful Doctor! Don't let your guard down. The law hasn't changed. Check before you chat—Think before you talk, and above all—get that release from your patient.

## TOPICS OF *Current Medical* INTEREST

### DX, RX, AND DRS.

By GUILLERMO OSLER, M.D.

Mrs. Royal Rudolph is the president of the WOMAN'S AUXILIARY to the Arizona Medical Ass'n. If she were asked to urge the women members to read ARIZONA MEDICINE, is COULD start a whole sequence of events,—the women members would read the journal; they would note the rarity of papers by physicians from Arizona; their husbands would then hear about it at home; more surveys and pieces of research would be started; and ARIZONA MEDICINE would eventually get more papers written by home talent. . . . Thank you, Mrs. Rudolph.

The pattern of susceptibility which allows so many young American men to have CORONARY ARTERY DISEASE has been worked out in the M.G.H. in Boston, under the guidance of Paul White and summarized by Gertler. . . . Individuals who are likely to have myocardial infarction can probably be identified well in advance of such illness. . . . The formula contains data on body build, physiology, personal history, heredity, and sociologic status.

Literature in favor of TUBERCULOSIS CASE-FINDING programs in the United States has made much of the fact that the disease is sometimes not officially known until death. . . . The publicists never had such good ammunition as the Arizona figures. The public health authorities first hear about at least 55 per cent of the tuberculosis cases when the death certificates are filed. . . . This column has pleaded for better reporting of the disease, and does so again. If a physician knows of a case, and if the patient is infectious and in uncontrolled contact with others, and if no report is made, it is akin to homicide with an accessory before the fact.

The Minnesota Medical Journal reports that the Hennepin County TB Ass'n. has started a campaign to have all Minneapolis hospitals X-RAY ALL NEWLY ADMITTED PATIENTS. . . . Very laudable. The paradox would seem to be that

J. Arthur Myers has almost convinced people that Minnesota doesn't HAVE any tuberculosis. . . . Maybe the survey is aimed at Loeffler's syndrome or Paragonamus Westernmanii.

SWELL SMELL QUELLS have been announced by two responsible companies. 'Odorant' is a 3½ watt lamp which can be placed in a wall fixture, and which is said by Westinghouse Electric Corporation to change the odor molecules of cooking, smoking, dampness, perspiration, etc., by the output of ozone. . . . 'Aero-Klenz Gel' is sold by the American Hospital Supply Corporation. It is said to deodorize putrid lesions for eight hours or more, and to be non-irritating.

More data are now available (in 'Anaesthesiology') on the narcotic DROMORAN HYDRO-BROMIDE. The drug was mentioned here a year ago. . . . It was used in 1,500 patients as a pre-operative medication, usually with atropine or scopolamine. The effect was unsatisfactory in 6.5 per cent, but almost all of these were due to lack of experience in dosage. . . . Nausea was very rare (1 case); pain and anxiety were absent; euphoria, hallucinations, and disorientation were not noted; and the patient could respond tho drowsy. . . . Sounds good so far.

The names of the ASSOCIATES IN GREAT MEDICAL DISCOVERIES often slide into limbo, except perhaps to people closely concerned. . . . Who, for instance, are Chain and Heatley? They were colleagues of Florey at Oxford who helped purify and first use penicillin. . . . Who were/are Slocumb and Polley? More familiar, but not as well known as their chief Hench, when they all demonstrated the use of cortisone and ACTH. . . . Then there is the man who refused to stay unrecognized, and sued Dr. Waksman, whom he helped discover streptomycin. So we've forgotten his name entirely!

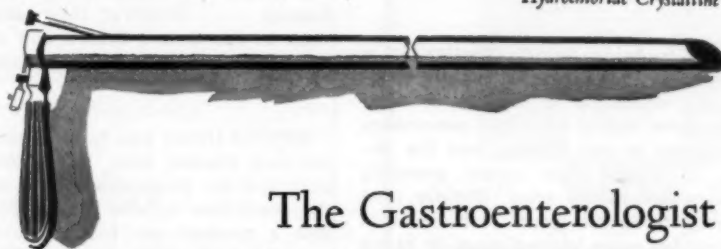
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The Danes report a new antibiotic 'LEOCIL-LIN', which has a marked predilection for lung tissue. It is a form of penicillin (to be precise, benzyl-penicillin-beta-diethylamino-ethylester hydriodide). . . . Ten times as much of the drug may be found in the lungs as of penicillin, tho the other organs have similar amounts. . . . It is excreted in the sputum, and naturally could be of great value in lung and bronchial infections.

**TERRAMYCIN is being found effective in a variety of conditions. It is said to be rapidly effective in whooping cough. It is said to be very helpful in ulcerative colitis (tho one remembers the psychic element in the disease, and the obstacles which a chronic case would present). Cholera is susceptible to the drug. Finally (and most importantly to brook trout) terramycin is the only agent which cures 'ulcer-disease' in brook trout.**

Chest specialists are hearing from their patients about a new anti-TB drug from Sweden. Now they may hear it from here. . . . 'PROTAP-TIN' was discovered by Davide and Theorell, and reported in Rome in June 1951. It has the same build-up as neomycin, viomycin, et al. Very good in cultures and animals, but we'll have to see later. . . . Could be used for the high incidence of streptomycin resistance in admissions to some V.A. hospitals (50%).

A question was asked in this column two years ago about the possible **ADDITION OF AN EME-TIC TO SLEEPING CAPSULES**. . . . Such capsules, containing ipecac with the barbiturate, have been on sale for two years. . . . They have been disapproved, however, since ipecac itself is toxic, and since the absorption and action would be slow enough so that too much sedative would also be absorbed. . . . J.A.M.A. Queries and Minor Notes now mentions that zinc sulfate is a better drug than ipecac. It works faster in producing emesis, and is non-absorbable. . . . Perhaps the objections to this idea will come sooner than 2 years.

**What a Long Tail Our Cat Has.**—Ideas for water saving, water production, water importation, etc., continue to appear. So far they are not yet enough. . . . 'Tax Talk', a Los Angeles publication, totals the cost of Colorado River water, Flood Control, and Sanitation Districts. It

is huge. . . . What interests Californians also interests Arizonans, and the plea of Tax Talk is not for more water sources but **DOUBLE USAGE** of what they have. How can used clean water from households be returned to the ground instead of being channelled to the sea?

A member of Alcoholics Anonymous has certain select qualifications,—he must have been an alcoholic, he must be cooperative, and he must be the type who responds to the religious appeal. . . . Consider how specialized a person must be who attends the **SECOND INTERNATIONAL Conference of PHYSICIAN MEMBERS of ALCOHOLICS ANONYMOUS**, at CLAYTON, New York, in August. . . . However there are 200,000 physicians in the United States, and more than a few have been known to take a drink.

**BERYLLIOSIS** has been a recent sensation in the 'new disease' field. The greater use of beryllium and the bizarre aspects of the lesions makes the conditions notable in medicine. . . . Now we find a medical use for beryllium, as described in a Machlett bulletin, the 'Cathode Press',—A new x-ray tube, containing a beryllium window, is made by them. It produces low-voltage radiation, and many superficial lesions are amenable to such x-rays, since they may be confined to a small surface area.

A new **CANINE ANTIRABIES VACCINE**, is 'strictly for the birds', but it eventually 'goes to the dogs'. . . . This paradoxical pun arises from the fact that the vaccine is 'avianized' or produced by growth in chick embryos. Contrary to the usual vaccine, it contains a live attenuated virus, and its effects also seem to last longer than the usual one year. . . . It is designed for prophylactic use in dogs, the species which is the major vector. . . . The U. S. Bureau of Animal Industry has released it for a controlled usage, and California State Health Department has distributed almost 3,000 doses.

## ANNOUNCEMENT

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### S.1186 AND H.R. 3298

*Statement of Dr. Walter B. Martin on Behalf of the American Medical Association Regarding S.1186 and H.R. 3298, 82nd Congress Before a Subcommittee of the Senate Committee on Labor and Public Welfare.*

Mr. Chairman and Members of the Committee:

My name is Walter B. Martin. I am engaged in the active practice of medicine in the state of Virginia and am a member of the Board of Trustees of the American Medical Association. I am appearing here today on behalf of that Association regarding S.1186 and H.R.3298.

Before proceeding, I would like to express the appreciation of the American Medical Association for the privilege of presenting a statement on these bills.

The substitute version of S.1186 as introduced on July 5, 1951 is designed to accomplish several purposes. The bill would amend the Federal Food, Drug and Cosmetic Act by expressly writing into law, provisions dealing with the labeling and dispensing of drugs which may be sold on prescription, and drugs which may be sold "over the counter." The Administrator of the Federal Security Agency would be given the authority to determine which drugs are unsafe and ineffective for use without professional diagnosis or supervision and thus to classify drugs into two groups on an arbitrary basis. The bill also provides that a qualified prescriber of drugs may telephone a prescription to a pharmacist, provided the pharmacist promptly reduces the prescription to writing and files it. If the original prescription was for a drug determined to be safe and effective for lay use without medical supervision, the bill authorizes the druggist to refill the prescription without confirmation from the physician. However, as to drugs which are habit forming or which are safe and effective only after medical diagnosis and supervision, the bill prohibits the refilling of prescriptions without expressed written or oral authorization by the prescribing physician.

On June 14, 1951, the Board of Trustees of the American Medical Association expressed their approval of the majority of the objectives sought by this legislation. This position was reaffirmed by the Executive Committee of the Board on August 4, 1951. The Association sup-

ports the provisions of the bill authorizing the filling and refilling of oral or telephone prescriptions. We also approve the restrictions pertaining to prescriptions for dangerous drugs. However, the Association is unalterably opposed to the provisions of the bill which give the Administrator of the Federal Security Agency dictatorial power to decide which drugs must be sold only on prescription and which must be labeled for "over the counter" sale. The delegation of such authority to the Federal government is believed to be extremely dangerous and wholly unwarranted.

It has been estimated that if this bill is enacted, it will be necessary for the Food and Drug Administration to promulgate regulations classifying over 30,000 drugs, and thus to specify the manner in which they may be dispensed. The necessity for continuous reference to such a list and to current official regulations in this regard would place a tremendous burden on the average druggist. The immensity of the task would inevitably result in technical violations, of no public health significance and would probably require the addition of numerous enforcement officials to police the procedure.

A more basic objection to the provision in question is that it vests in the Federal Security Agency, control of the drug industry and delegates to that agency the power to determine the therapeutic value of drugs—a decision which is a traditional and time tested function of the medical profession. In this respect federally placed control of the drug industry, especially as to the effectiveness of drugs will result in unnecessary and undesirable control of the practice of pharmacy and the practice of medicine.

There is also pending before your Committee another bill, H.R.3298, which was passed by the House of Representatives on August 1, 1951. This bill was amended on the House Floor so as to strike out the provisions which would give the Federal Security Administrator power to classify prescription and non-prescription drugs, to which our objections are directed.

The American Medical Association favors the enactment of H.R.3298 with one minor amendment. It is suggested that a period be placed after the word "sentence" on page 3, line nineteen, clause (b) (4) and that the following words beginning on line 19 and continuing through line 22 be eliminated: "or any other





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statement which represents or implies that the dispensing of the drug without the prescription of a licensed practitioner is prohibited." It is believed that this wording is ambiguous and unnecessary. The prohibition against the improper use of the caution legend included in the same sentence should provide a sufficient safeguard.

Again let me thank you for affording us the opportunity to present these comments.  
September 13, 1951

### REGIONAL MEDICAL MEETING

The Coconino County Medical Society joined the Medical Societies of the five Northern Counties Wednesday, October 3rd, in a Regional Meeting at the Bright Angel Lodge at Grand Canyon. The meeting, organized by Dr. Leo Schnur, President of the Coconino County Medical Society, had as its general theme the topic, "Psychotherapy in General Medicine."

Registration was held in the morning, followed by luncheon. Dr. Harold C. Bryant, Superintendent, National Park Service, Grand Canyon, delivered a welcoming address to the visiting doctors and wives.

Guest speaker was Dr. Hale F. Shirley, Associate Professor of Pediatrics and Psychiatry at Stanford University and Director of the University's Child Guidance Clinic in San Francisco. Dr. Shirley stressed the importance of the non-psychiatric physician's role in dealing with the emotional aspect of his patient's illness. Dr. Clarence Salsbury, of the Arizona State Department of Public Health, acted as moderator and led the group in general discussion following Dr. Shirley's remarks.

Dr. Henry C. Schumacher, San Francisco, Medical Director of Region 10 of the U. S. Public Health Service, also spoke to the group.

Notable among the physicians present were Dr. Dermont W. Melick, President of the Maricopa County Medical Society; Dr. Charles W. Sechrist, Flagstaff; Dr. Herbert R. Rice, Flagstaff; and many leading doctors from most of the counties in the state. Mr. William B. Macomber, Supervisor of Mental Health, State Department of Health, Phoenix, and Miss Courtney Rettger, Tucson School System psychiatric social worker, contributed remarks to the general discussion.

The doctors' wives were entertained at bridge during the afternoon at the El Tovar Hotel.

The medical group enjoyed a social evening following dinner at El Tovar.

### SCHOLARSHIP IN PSYCHIATRIC NURSING

Word has been received from the ARIZONA FEDERATION OF WOMEN'S CLUBS that a \$500.00 Scholarship in Psychiatric Nursing is now available to a graduate nurse who will give the benefit of this training in Arizona.

This program of the Arizona Club is part of a National Program sponsored by the GENERAL FEDERATION OF WOMEN'S CLUBS. Facilities of Louisville's Norton Memorial Infirmary Psychiatric Clinic, in Louisville, Kentucky, are being made available to nurses from all sections of the nation for postgraduate psychiatric nursing education. The Norton Psychiatric Clinic is recognized by the Division of Nursing Education of Columbia University, New York City. Under provisions of the National Mental Health Act, there are provided clinical instructors for teaching psychiatric nursing, and resident fellowships for doctors completing their formal psychiatric education.

Nurses will be accepted for instruction at intervals of two months. The course, centering in the Norton Psychiatric Clinic, is amplified by coordinated training at Louisville General Hospital, the Mental Hygiene Clinic, and the Child Study School.

For additional information and application forms, contact:

Mrs. Hazel D. Smith, Scholarship Chairman  
Arizona Federation of Women's Clubs  
501 East 16th Street.  
Tucson, Arizona

### PERSONAL NOTES

*Dr. Henry A. Siegal*, 15 East Monroe Street, Phoenix, Arizona, has recently returned from Chicago where the degree of Certified Fellow of the International College of Surgeons was conferred upon him.

*Dr. and Mrs. Kenneth C. Baker* have returned from El Paso where they attended the meeting of the Southwestern Dermatological Society which convenes in conjunction with the Southwestern Medical Association. The meeting was held at the Hotel Cortez Saturday and Sunday, October 20 and 21.

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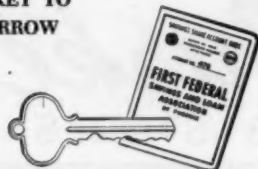
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## AMERICAN CANCER SOCIETY

A statement of the aims and objects of The American Cancer Society, Arizona, Division.

The American Cancer Society, through its representative organization, the Arizona Division, solicits the cooperation and support of all physicians in the State of Arizona. As is logical, the primary aim of the American Cancer Society is to discover, through research, the cause of cancer. It is proper that the greatest effort of The American Cancer Society should be in the direction of research. Until the day when cancer can be eliminated the American Cancer Society stresses, as its second great aim, the early recognition of cancer so that it may be adequately treated. Its educational program, therefore, embraces all aspects of the cancer problem from cancer education at the physician and medical school levels through cancer education of the general populace down through the school child. With that in mind every medium is used and the general population has become aware of the so-called "Seven Danger Signals". The importance of these two major programs becomes apparent as cancer rises in the list of causes of deaths to a higher and higher position, not only because of the elimination of infectious diseases and other previously more significant causes, but also because more people now are entering the older age groups where cancer is more prevalent.

The third phase of the cancer program is the service program. This includes the provision of financial aid and assistance to those suspected of having cancer who require diagnostic studies and hospitalization and in some instances assistance in proven cases of the disease. Dressings are provided when necessary and the program includes the maintenance of such detection centers as are operated in our area. Of necessity the service program is limited in its scope for the funds available to the American Cancer Society are in no way comparable to those raised by other similar health organizations. The service program in its application is offered to those individuals who are unable to pay for similar services in the usual manner and who are not eligible for care in county hospitals, governmental institutions or through assistance from families.

It must be apparent from the above that the first and second aims of the American Cancer

Society should be given preference. The service program must be curtailed because of lack of funds. In actual operation requests for service help should indicate the name of the attending physician or recommendation of the cancer clinic or detection center. Such requests for assistance should be directed to The American Cancer Society, Arizona Division, at 25 East Van Buren Street, Phoenix, Arizona, or to the local representative of that society in each county. In general social service information and welfare records give information to confirm that help is being given to needy individuals, and such a survey is necessary to assure the proper distribution of help. Service funds can be applied in amounts usually limited to \$75 or \$100 per case, with a few exceptions, and these funds are paid to hospitals or diagnostic organizations upon billing. Physicians have universally offered their services to such indigent patients without charge.

It is the hope of the Professional Board of the Arizona Medical Association that each doctor's office throughout the state will become the primary initial detection center. Cases suspected of having cancer and requiring help from the American Cancer Society it is hoped will be promptly cared for. Thus the American Cancer Society will have the opportunity to contribute to the early recognition and proper treatment of all malignancies.

## ARIZONA HEART ASSOCIATION

The annual meeting of the Arizona Heart Association was held in Phoenix, Arizona, Saturday, September 29, 1951, at Hotel Westward Ho. Following opening remarks by Benson Bloom, M.D., Tucson, Program Chairman, John C. Jones, M.D., of Los Angeles, Associate Professor of Surgery, University of Southern California, discussed "Congenital Heart Surgery." His address was followed by Louis N. Katz, M.D., of Chicago, Illinois, President of the American Heart Association, who discussed "Recent Trends in Clinical Electro Cardiography," and "Recent Developments in Atherosclerosis." John C. Jones, M.D., presented further discussion on "Mitral Valve Surgery."

Dr. Clarence L. Robbins of Tucson succeeds Dr. Kent H. Thayer of Phoenix as President of the Heart Association and will lead this Association's efforts to raise \$30,000 in a campaign to be

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inaugurated in February, 1952, for the fight against heart disease. Dr. Robert S. Flinn of Phoenix was named President-Elect to succeed Dr. Robbins next October. Other officers for the coming year are Dr. Ben P. Frissell of Phoenix, Vice President; Robert H. Whitacre of Phoenix, Vice President of the First National Bank of Phoenix, Treasurer; Miss Elizabeth Rockwell, Secretary; and George F. Stoeberl, Executive Director.

### FROM SECRETARY LULL'S LETTER

*Tells What Makes A.M.A. Tick.* President John W. Cline gave an excellent and most interesting address before the annual session this week of the Medical Society of the State of Pennsylvania at Pittsburgh. His title was "You Are the A.M.A."

He recounted the part which Pennsylvania physicians have played in the growth of the A.M.A. and then pointed out some of the responsibilities which doctors have toward their national association.

"The responsibilities," he said, "are yours because YOU are the A.M.A. As an individual, as a county medical society member, as a state society member . . . YOU are the A.M.A. You elect its officers, promote and implement its program, and finance its activities through your annual dues. You share in its accomplishments and, by the same token, you must share the criticisms levied against it.

"In an organization as large as ours, it is regrettable that so few do the work for so many. How many times have you heard your minister preach on church attendance? Invariably, he says that, to his sorrow, the members of his flock he really wants to reach are the ones who aren't in the church.

"In the same way, I can say that the majority of the physicians here today are the workers . . . the men who constantly give their support and assistance to the A.M.A. and their county and state societies. For that reason, this meeting can be more accurately classified as a sales conference. You are the A.M.A., and it's up to you to carry the information you receive today back to the hundreds of physicians in Pennsylvania who don't attend state meetings."

Dr. Cline listed several specific criticisms leveled at the A.M.A. and the medical profession, and told what should be done to correct

the trouble. He concluded by saying that "every physician should work closely with his national organization to correct the misunderstandings that prompt criticism of our profession."

"Unfortunately," he added, "the basis of most misunderstanding either begins to germinate or could be clarified during the initial visit of the patient to his doctor. You are the A.M.A. to your patients and whatever they think of our association . . . and our profession . . . will depend on your personal responsibility as a good will ambassador."

Dr. W. W. Bauer, director of the A.M.A. Bureau of Health Education, spoke at the same session. The title of his talk was: "What Do You Get For Your \$25?" He did a fine job of explaining the many services which are available to physicians through their national association.

At about the same time, Dr. Edward J. McCormick of Toledo, a member of the Board of Trustees, gave a talk very similar to Dr. Cline's before the annual session of the Colorado State Medical Society in Denver. His speech was also entitled "You Are the A.M.A."

Dr. McCormick said that he was willing to wager that "if someone stopped you on the street and asked, 'Say, Doctor, just what is the American Medical Association?', you'd come up with a long explanation about our Chicago headquarters and our hundreds of activities."

"To my way of thinking," he said, "you'd be wrong. The A.M.A. isn't a big stone building in Chicago; it isn't a bunch of councils and committees and bureaus all lumped together. Doctor, the A.M.A. is YOU. You are the A.M.A."

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Front Row: Seated, Shirley Koluch, Phoenix, Arizona; Joan Nasser, Miami, Arizona; Frances Lopez, Morencí, Arizona. Back Row: Standing, Mary Gustafson, Tucson, Arizona; Jackie Johnson, Phoenix, Arizona. Mary Gustafson is in training at Good Samaritan Hospital. The other girls are at St. Joseph Hospital. These are all the student nurses in training in Phoenix. There is one in Tucson who is not shown here.

### STATE NURSES' LOAN

To the doctor's wives in the state of Arizona the Nurses' Loan Committee of the Auxiliary to the Arizona Medical Association makes a direct appeal for help and co-operation. The Nurses' Loan program is beginning its second active year, but it needs the assistance of all doctors' wives to publicize the program. Will you, after you read this article, go to the Clubs and various organizations in your community and explain the program? Also, will you go to your high school principals, high school nurses, and vocational directors and tell them of the availability of the loan for worthy girls who wish to become nurses? We have sent letters for the past two years to all high school principals in the state telling of our loan fund, but we know that the personal approach brings better

results.

Here is a list of our requirements:—

1. The applicant must have a B average
2. The applicant must pass a strict physical examination
3. The applicant must *need* the loan
4. The applicant must be of good moral character and must furnish three character references, one of which must be from a member of the high school teaching staff
5. The applicant must have a parent or guardian co-sign with her to agree to repay the loan. The maximum loan granted is \$300, interest free. The applicant must agree to repay the loan at the rate of not less than \$12.50 per month. She must start repayments within six months after she receives her state license to work.

If she does not finish training, the applicant must agree to start repayment of the loan within thirty days after leaving the School of Nursing

6. The applicant must be accepted by one of the four accredited Schools of Nursing in Arizona. These are:—St. Joseph's Hospital, Good Samaritan Hospital, Memorial Hospital (all in Phoenix) and St. Mary's Hospital in Tucson, Arizona.

Last year the Nurses' Loan Committee gave financial help to four girls. This was the first year of the program. We gave loans to two girls in training at Tucson, and to one girl at Good Samaritan Hospital in Phoenix. This girl was second highest in her class. We also gave a loan to a girl at St. Joseph's Hospital. This fall the Committee voted to give loans to three girls, one from Morenci, one from Miami, and one from Phoenix. They entered St. Joseph's School of Nursing on September 19, 1951.

Pima County Auxiliary will have a large tea in October and will invite junior and senior girls who are interested in becoming nurses to attend. Maricopa County probably will have a similar function. Please inform your Community of the program so that the Auxiliary can contact and help more worthy girls to become nurses.

Respectfully,

Josephine B. Craig

(Mrs. Carlos C. Craig)

Chairman State Nurses' Loan Committee

## GETTING THE MOST FROM YOUR BULLETIN

The Bulletin, well known as the official publication of the Auxiliary, gives to the membership information and suggestions that will be of interest to them and help them in their work. In order to keep informed and up to date, one must acquire the habit of reading the publication.

From the pages of the August issue let me acquaint you with some of the worthy projects accomplished in other states.

In California, Civilian Defense is a vital new project which has been added to their agenda. Auxiliary members have been working as individuals in various community capacities, but as auxiliaries have been requested to make master files of all nurses, registered and practical, with

pertinent information as to training and availability. One county sent questionnaire cards to all members to ascertain abilities, training and interests in connection with Civilian Defense.

The inadequate care of the mentally ill is by far the greatest health problem in the State of Florida. A State Mental Health Society was organized and a member from the Auxiliary executive board was appointed legislative chairman for this committee. Information concerning present conditions with proposed legislation for the correction of same were sent to every organization throughout the state. The legislative chairman of the Auxiliary has worked with the Mental Health Society in obtaining public support of proposed legislation. There is a definite awareness among those with political ambitions of the existence of a medical Auxiliary in the State of Florida.

Nurse recruitment has been a major project for the State of Kansas. Most of the auxiliaries included a program on nursing. In 15 counties, 22 scholarships have been developed, 12 counties sponsored programs in high schools to interest girls in nursing, and 10 organizations presented films on nursing to all the high schools in their counties.

Louisiana's nurse recruitment program has expanded due to the outstanding work of the medical auxiliary. The schools of nursing showed the highest percentage increase in admissions of new students of any state in the country last year, with a 53% increase over 1949 and a total of 741 first year students. Programs varied throughout the state. One auxiliary had a window display in a leading department store, while another depicted the story of the shortage of nurses in a Junior Chamber of Commerce "Democracy versus Communism" parade. Literature on nursing and against compulsory health insurance was distributed to thousands of on-lookers.

Kentucky became aware that most of the college and university editors in this country were receiving from various sources much socialistic propaganda to be used either in the student publications or as material for programs on the campus. In order to offset this propaganda the auxiliary sponsored a college Editors' Contest on Americanism in senior colleges in Kentucky. Every editorial staff of every senior college was visited personally and the purpose

of the contest was explained to them. They felt that in this contest "seeds of inspiration were sown in the minds of many college students who might otherwise have been indifferent."

"Health Days", of which Minnesota is so justly proud, continues to be their greatest contribution to public relations. These are sponsored jointly by the State Auxiliary, the State Medical Association and State Department of Health. Programs are made up of panel discussions, films concerning cancer, tuberculosis, accidents in the home and health programs concerning schools. Sanitation and legislation are discussed. Many county auxiliaries have "Guest Days" to which other organizations are invited to hear the best informed speakers on pertinent subjects

The activities in these states are but a few of possible lines of endeavor which may absorb the workings of the auxiliary. There are many others and we should surely never want for lines of achievement in our own auxiliary. Our organization is continually becoming more active and it is hoped that our future projects may be as well chosen and successful as the above-mentioned ones have been.

Irene R. Hewitt (Mrs. Roy)  
State Bulletin Chairman

#### H.R.-910

### FEDERAL AID TO NURSE EDUCATION

This bill provides federal aid to nursing education in four different categories:

1. Annual payments to schools for cost of instruction
2. Grants for construction and equipment of new schools or expansion of existing schools.
3. Scholarships.
4. Grants for a recruitment program.

Both proponents and opponents generally agreed that a shortage of nurses or nursing services exists but opinion varied as to degree, nursing schools are short of instructors and a proviso for a court appeal in the event funds are withheld by the government should be written into the bill.

Dr. Walter B. Martin, of Norfolk, Virginia, a member of the Board of Trustees of the American Medical Association, appeared before the interstate and foreign commerce committee to express opposition to many parts of H.R. 910.

The Association objected to intrusion of the federal government and its agents into fields of education whether it be general education, medical education or nursing education. This bill would give the Surgeon General of the Public Health Service, under the Federal Security Administrator almost unlimited administrative powers. Also with a system of subsidies established it will be difficult or impossible to terminate them once the schools have become dependent on a federal dole. Their sources of private support will wither, and their freedom will be lost because they will necessarily have to comply with every federal regulation or go out of existence. The American Medical Association feels that there is a present shortage of nursing service, although there is not an actual shortage of nurses. Of the 500,000 trained nurses in the United States there are approximately 300,000 who are following their profession, and the reserve of 200,000 nurses could be tapped in the event of a national emergency.

Dr. Martin proposed the following in lieu of H. R.-910:

1. One-time construction grants on matching basis, based on formula similar to that of the Hospital Construction Act. (Hill-Burton Bill).
2. A grant of federal funds to Committee on Careers in Nursing or some comparable private agency to help support a nurse recruitment program.
3. A temporary grant-in-aid program, not exceeding five years, of scholarships for advanced nursing education, administered by states.

A three point program of this type should provide for the construction of necessary additional nursing schools and the renovation of certain existing facilities; for a strong national promotion and information program to bring qualified young people into nursing, to improve the profession and encourage nurses to remain active in their profession; and for an increase in the number of qualified teachers in the nursing field. It is believed that these suggestions, if put into effect, would meet the current need and at the same time safeguard the freedom and independence of educational institutions far more satisfactorily and specifically than does H. R.-910.

Mrs. Louis Hirsch  
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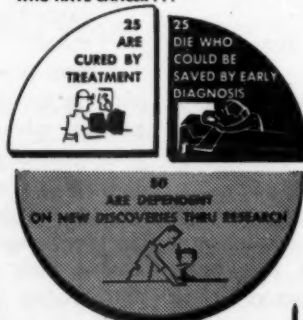
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\*Based on average reported values for milk.

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